VOLUME LX APRIL, 1904 No. 4

AMERICAN JOURNAL OF INSANITY

THE DEVELOPMENT OF INSANITY IN REGARD TO CIVILIZATION.

BY ROBERT JONES, M. D., LOND.

B. S., F. R. C. S. Eng., M. R. C. P. Lond.

Resident Physician and Superintendent to the London County Asylum, Claybury. President of the Section "Psychological Medicine," British Medical Association.

It is a universal law that the most highly integrated functions of vital organisms are the most apt to suffer derangement, and the object of this address is to demonstrate that, with the progress of civilization, mental breakdown becomes more serious and more frequent, and the varieties of insanity are more chronic and less curable than in the early days of a century ago, when life was simpler and men were more content.

The human mind—the most highly evolved process in relation to matter—has from the earliest times been subject to some obscuration or disintegration, but the cure and care of the insane, now so much in the "ambient air," was hardly known as a subject of serious study a hundred years ago. The last century has been more marked than any of its predecessors in regard to the material, mental and moral progress of mankind, and a short review of this progress with some lessons to be learnt from it cannot but be a matter of much interest to practical persons. Over many of us the history, the traditions and the personal narratives of the past cannot but exercise a potent spell. We can learn from our failures as well as from our successes, and these failures may possibly convey to us some notes of warning and act towards us as beacons and signals of danger. As,

however, "hope springs eternal in the human breast," and our faith and aspirations are ever forward and to the future, some hopeful prophecy might be expected as to the alleviation, if not the prevention, of mental derangement; but will the lesson of our retrospect justify this anticipation?

The sum total of the physical, moral, social and religious energy which has caused our progress during the past century has been unparalleled in the history of man, but action and reaction are always equal and contrary, and we shall find that our advance has not been without sacrifices—losses here and retrogression there—and that civilization itself has not been an unmixed blessing, for in the struggle which has marked our advancement, the conflict between intelligence and ignorance has resulted not solely in pure reason and limpid wisdom, but the path of progress has been freely strewn with mental wreckage and physical degeneration.

In the whole range of medical science, insanity is, perhaps of all others, the most painfully interesting subject. It is the debatable land of the imagination, which presents many subordinate varieties of its wanderings, the dream of the poet, the fable of the mythologist and the fiction of the romancer being woven with its threads.

Insanity, in the excursions of mania with its varieties, its fancied consciousness of unlimited power, its self-satisfaction—that abundant source of mental delight—bestows upon its victims feelings of bliss much more exalted than fall to the lot of sober reason, but insanity in the self-condemnation and misery of melancholia inflicts far severer pangs upon its victims than can be produced by the most extraordinary anxiety and the most acute bodily pains. What are the consequences of insanity? It means in the first place a deprivation of liberty, perhaps a lifelong maintenance out of the public purse, already heavily overburdened; a maintenance including housing, clothing and food which tends each year to rise in cost. In addition to this useless life, or a living death, a stigma probably attaches to those who are related to and are dependent upon the sufferer, by which they are made to feel the curtailment of the necessaries of life, increasing the liability to a similar breakdown in themselves. Surely, if the possession of reason be the proud

attribute of humanity, may not such a disease of the reason as is insanity be ranked as the greatest affliction, and is one not forced to make the study of such a disease which touches society, involves the law and defies medicine, one of deliberate and anxious thought?

What is it that characterizes insanity? It is not the presence alone of delusions or hallucinations. There are many persons in daily life who fulfil all their duties and obligations to society and themselves, and yet suffer from hallucinations. Insanity, although a perversion of mental functions, is not exclusively an intellectual disorder, for persons gifted with high intelligence may fail to respond to ordinary motives, and become so defective in their habits as to be socially unfit to mix in the world. Again, the distribution of mental faculties may be so uneven and mental endowment may be so unbalanced that their possessors are unfit to be at large. There are many persons who are properly incarcerated in asylums, in whom there are merely exaggerations of normal tendencies, fluctuations of the mental faculties which may be described only as excessive or beyond normal limits and which render these persons unstable, untrustworthy and even dangerous, yet in whom there is hardly, if any, loss of mind. It is only too well known that there are imperceptible gradations as well as abrupt transitions between health—which is the easy, harmonious and unconscious performance of the organic functions—and disease. Health of mind, like health of body, is a somewhat indefinite expression.

I shall exclude from the subject of this paper senile forms of insanity, the mental decay of involution, which results in more or less normal loss of mind, as also the primary defects of idiocy and imbecility, which are pathological variations not entirely dependent upon individual stress, and which exist in all grades of society and all states of civilization.

So far as any trustworthy information is to be had it appears that insanity increases as man departs from the savage and semi-civilized states and approaches the highest civilization. In primitive states of society and among uncivilized races, insanity is rare, the pure psychoses are unknown; the chief forms—apart from the low grades, idiocy and imbecility—being associated with the taking of drugs and corresponding to the insanities of

civilization resulting from alcohol, ether, cocaine, morphine, etc., and which in the main are curable. Insanities which end in dementia are rare, and dementia itself is uncommon, and it is only when we come to higher civilization that the higher forms of insanity are seen. Diseases, like the physical causes which produce them, have their evolution, their cycles, and their day, and I shall proceed to demonstrate that the progress of mankind has caused a more or less complete change in the type of insanity such as now comes under treatment. As to the causes of insanity, the actual and proximate exciting causes will always be stress of some kind acting upon an organism predisposed to breakdown, the great predisposition being determined by heredity and by the social environment of the individual, for we know that social relations and obligations constitute the preponderant factor in any mental life. As to heredity, we need say no more than that an inherited instability of organization makes itself evident in the present day more than in any previous period at the physiological crises of life, a hereditary tendency being responsible for more than one-third of all occurring insanity. The tendency of recent years to look upon insanity more as the result of bodily than mental causes is no new view. Haslam, in the early part of the last century, classified the causes of insanity and insisted that the disease was never purely mental; that, on the other hand, positive bodily conditions were the causes and that insanity was structural and active in its origin. Earlier writers, however, laid great stress upon a disturbance of the emotions as a cause of insanity, sudden violent affections, or the habitual indulgence of any passion whatsoever being looked upon as a definite exciting cause, those whose professions required the exercise of the imagination and the indulgence of the passions, such as the *personnel* of the drama, being said to be peculiarly liable to insanity, although probably some physical factor, such as a dissolute and irregular life, was concerned in causation. Fright, anger, religious terror, grief, ungratified desire and disappointed pride were specially referred to by these earlier writers as definite causes of insanity. It was pointed out by Pinel that 30 per cent of all his cases were due to domestic bereavement, 26 per cent to the events of the Revolution, 22 per cent to fanaticism and disappointment in love. It is also an interesting record by Charles

Booth (Life and Labor of the People) that the anxieties and uncertainties of professional life are responsible for the largest proportion of insanity which this class of all others shows. He also points out that loss of trade affecting bodily health is responsible for causing insanity, and that provided wages are regular, although low, and there is no nervous strain, there is little tendency to insanity.

As to civilization, this means progress, and it implies a development from the simple to the more complex. When the conditions of life become more specialized the nervous system of man has to react to more stimuli; not only are these more numerous, but they are more complicated. For each stage of civilized life a special standard is fixed, and the more perfect and the higher the standard the more culture and knowledge are attained and better results obtain in consequence. Highly civilized man thus lives in a more complicated environment, which calls for the higher forms of self-control and more prolonged and varied efforts than are customarily aroused by the simple emotions and the elementary sensorial stimuli of a primitive life. In these efforts numbers of unfortunate ones will not succeed; they are incapable of elevation to this higher plane of civilization owing to mental, physical and moral deficiencies. Not having the qualities essential to success, they are left behind, evolution proceeding at the expense of the less fit—those, in fact, whom civilization itself in the struggle renders unfit for the standard it has itself fixed. It is thus seen that civilized society, in forcing the pace, practically manufactures its own unfit, viz.: its own paupers, its own lunatics and its own criminals. London alone in this respect is responsible for the production of over 70 insane persons per week, and this number is destined unrelentingly to increase. The only neutralizing agency is the fact that sterilization follows in the wake of three or four generations of town-bred people, a natural law which ensures that the unfit shall cease to cumber the earth. Not that civilized life, properly developed, tends in itself to cause insanity, but by implying a more complex mental structure with more delicate parts and nicer construction, this mental machine is more liable to faults of mechanism and varieties of derangement than is the case with the simpler and more vacant minds of primitive people. In cities where the population has

to accommodate itself to the pressure of competition, the tension of mind is more continuous. There is an increased inhibition from pent up mental energies which in a primitive life find a ready exit through muscular actions. In cities, also, artificial desires multiply, unhealthy activities are created and ambition further forces the overworked brain, conditions which, sooner or later, are liable to cause its complete breakdown. The wants of modern civilized life are many and are rarely gratified, the eager hand reaches to grasp the prize which is plucked away by some other of the numerous competitors and bitter disappointment is added to the nervous strain and mental overwork. It is doubtful, however, if mental labor alone is ever an actual cause of insanity, for habitual application of the mind, when not excessive, strengthens it and renders it less liable to disease. Dr. Mott has shown that the best and most exact nervous reaction takes place when the nerve circle is complete and in a state of healthy tonus or strain. There is no doubt that fatigue is unfavorable to good mental work, and by experience we know that fatigue is caused by intense thought. Böerhäave said that unremitting mental attention to a single subject for a whole day once kept him for six weeks without sleep. Bodily fatigue we also know to be demonstrably injurious to thought, for Kraepelin has shown that muscular exhaustion weakens brain power in definite curves and ratios. It is not overwork, however, so much as worry and anxiety which cause insanity. Civilization, as we all know, brings in its train different effects to different groups of people. To the idle rich it brings sensuous luxuries of all kinds which account for the insanity of those in easy circumstances who are prone to much self-indulgence, who pay over much attention to personal longings and sensations, and who have too little occupation, or whose occupation is irrational and unhealthy. To the poor—the stratum in society most prone to insanity—who are at the mercy of every economic fluctuation, it brings lack of proper nutrition, overcrowding with unsuitable hygienic and moral surroundings, and all the other evils and abominations caused by the massing of people in large cities, such as alcoholic and other indulgencies, poverty, and crime, the latter an evil worse than poverty and which bears a very intimate relation to insanity, the instinctive criminal and the morally

insane being considered to be identical individuals, characterized by the same psychical and physical stigmata of degeneration. As to overcrowding, an immense number of poor people live in small and badly ventilated apartments with filth and squalor in their mean streets and dwellings. Their food is often deficient in quality and quantity, which predisposes them to physical and mental disease. How can vegetables, fish and milk be fresh and digestible by the time these reach the poor of London, where even the cooking for this class is stated on good authority to be worse than in the country? Surely the plainest food with health under a hedgerow must be better than semi-starvation with the glamour of the city. Overcrowding leads to many and various miseries and personal discomforts which are humiliating and demoralizing to the grown-up and are a source of moral contamination to their descendants, in whom they cause mental and bodily degeneration. All the year round these dwellers of the city are at a disadvantage when compared with the country folk In winter they suffer from cold, want of food and clothing, and in summer they endure a debilitating atmosphere from the reeking odors of dirt, decomposing garbage and noisome refuse. The disposal of refuse, the sterilization of milk, the detection of adulterations and the provision of pure water are great problems which big cities have ever before them. London, probably the healthiest of these, breeds her legions of anæmic, hydrocephalic and unhealthy children from the want of pure air and radiant light. Crowds of people have their happiness stifled by their environment in our great cities, with the result that they seek to drown their misery and depression in the effects of alcohol. This sedative, taken to lull and veil the little worries, the small pains and the general mental disturbance of whole classes, is the cause of at least a fifth of all insanity occurring in males and more than half this proportion in women—the proportions are probably much higher, and call loudly for definite action and treatment. Such indulgence leads to other sensuous excesses which are all deleterious to nervous centers. There is no doubt that London and, *a fortiori* other towns, produce in the present day a tension of the nervous system as baneful as it is unnatural. One has only to look at the living maelstrom which pours into airless and sunless London offices, workshops and fac-

tories every day from the suburbs to see the strained, eager, earnest and inwardly preoccupied people who are compelled to sacrifice their health and overstrung nerves in the cause of "civilization."

Let us briefly contrast the state of civilization a century ago with what it is to-day, and see for ourselves the reason why the stream of progress has been strewn with physical and mental wreckage. Beginning with education, the great mass of the people was entirely without it. At Queen Victoria's accession 44 per cent of the community were unable to read and write, or even to sign their names. Now this proportion is less than 6 per cent. Another great change within the past century has been the migration of the population from rural districts into towns and which has been caused, in the main, by our present system of education.

In the first year of Queen Victoria's reign sufficient wheat was grown on the land to feed the people for eleven months in each year; now there is only enough grown to provide the population for two and a half months. Furthermore, the cultivation of the potato—a measure of industrial as well as of arable progress—is declining in this country, where it is only 38 acres per 10,000 persons, as against 161 in Germany and 98 in France. In the year 1800 more persons lived in the country than in the towns; in 1841 the urban total was one-half of the whole population, and in 1891 the urban constituted 77 per cent of the whole population. Such a system of education which drives each year 80,000 persons from the country into the towns, and, as Rider Haggard states, leaves the land without laborers and ploughmen and which yet causes millions a year to be paid away to the little culturists of the Continent for fruit and eggs, and over half a million for onions alone, which can so well be grown in this country, is evidently unsuited for rural districts. Not until each local authority can determine what should be taught in each district, as foreshadowed in the New Bill, can any improvement take place. The present system of education is too rigid, and the hard and fast curriculum of cramming should give way to a minimum of bookwork and a maximum of outdoor instruction, so as to train the eye to observe and the hand to work.

Shelley's diatribes against the selfish oppression of the people

by the landowners and others were doubtless fully justified, but it is a question whether the political franchise has not extended power into the hands of masses of people who are incompetent to grasp issues involving great risks. What is now the condition of the villages, the looms of which were once in active work, and where the handicraftsman once throve in patient contentment, feeling joy and pleasure at the performance of work done as well as it could be done? These handicraftsmen have now left for the towns, where they work in small, hot and ill-ventilated shops, and are at the mercy of every trade fluctuation, the curse of drink following on the heels of their uncertain occupations.

Perhaps in no department of human activity has progress been more marked than in that of communication. Means of communication were rude and costly, and only the well-to-do could travel; roads were bad, tolls and turnpikes were frequent. The stage coach was the only conveyance for the many, but life in the open air tended to chivalry and courtesy. Our ancestors read less than we do, but their reading was of a more solid and enduring character than is ours. The scarcity of books and the comparative rarity of journals induced readers to master what they read, with the result that they absorbed real nourishing intellectual food. A superficial attention to an ill-digested course of reading dulls and benumbs the intellect. I have had, not one, but several youths whose insanity was distinctly traceable to the injurious tone of the literature they indulged in. It is also stated, by those best qualified to give an opinion, that this steeping of our youth in the prurient, exciting and unreal literature of to-day—a literature that crowns even vice and crime with an illusory halo of glory—has been the principal cause of neglected domestic duties, unhappy homes, and a large percentage of crime, as is frequently testified in the summing-up of our magistrates.

The emancipation of women from unfair and unjust disabilities, and the large measure of education and freedom granted to them, have especially marked the last century; but it is a serious assertion that they look with complacency upon the conjunction of an increased marriage rate and a diminishing birth rate. It is believed that the idle women of the upper

classes (who are more self-indulgent than ever before) regard the immorality of the average man with less aversion, and that they bewail the injustice of visiting condign social ostracism and punishment upon women for similar delinquencies. Further, the revolt against domestic service among the lower classes is also to a great measure a reflection of the love of pleasure in high places.

I do not deny that vast improvements have taken place in the social condition of the people in the past century. But what have been the physical and psychical effects of progress upon the nation? In spite of the progress in sanitation, the science of preventive medicine, which concerns the great tripod of life—food, air and water—and which now forms one of the most important departments of medical education, a science which in the late reign has caused 150 millions to be spent in providing pure water alone, which has directed special attention to temperance, to the reduction of mortality (the rate of which has declined from 1 to 23 in 1685, 1 to 40 in the middle of the century, to 1 to 55 at the present day), and especially to infant mortality—calculated by the proportion of deaths under 1 year of age to 1000 births—which, according to the Registrar-General's information, was yet higher in 1902 than 1881 (one-half of all children born dying before the age of 5 years), and to the protection of adult and child life by special statutes, yet I say that in spite of all these facts the physique of the nation has deteriorated, the deaths from cancer and nervous diseases have absolutely increased, as may be seen from the following table :

ANNUAL DEATH RATE PER MILLION PERSONS.

(*Spectator*, July 4, 1903.)

	1861-65	1886-90
Cancer	367.8	599.7
Nervous diseases	1546.0	1785.3

Furthermore, out of 1000 consecutive male cases admitted into Claybury Asylum, 143 were between the ages of 15 and 25, inclusive, and these are typical of their class. The following table gives the actual physique of these 143 persons in weight and height at the respective age period, compared with the averages in the general population at the same ages:

No. of Patients Admitted.	At the Age of	Average Height of Patients.	Healthy Standard of Height According to Age.	Average Weight of Patients.	Healthy Standard of Weight According to Age.
		Inches.	Inches.	Lbs.	Lbs.
1	15	56	61	64	103
6	16	61	63	109	117
4	17	64	65¼	117	130
12	18	65	65¼	120	142
16	19	64	65½	122	139
13	20	65	65¾	123	138
22	21	66	66	130	145
17	22	64	66¼	130	141
16	23	66	66¼	128	145
20	24	66	66¼	132	145
16	25	64	66¼	127	144

Does not the above point to a physical degeneration in certain strata among a large number of the population, for which some cause should be found? I know it is asserted that the growth of the upper classes has increased, but where is the proof of this? Talbot and Kingsley have recently called special attention to the contracted jaws and poor teeth of town-bred persons, and the attention recently called by the Royal Commissioners on Teaching in Scotland, by the report of the Inspector-General of Recruiting, who states that one out of every three persons brought up by the recruiting sergeant is rejected, by a special report of the Director-General of the Army Medical Department, by Sir Lauder Brunton and many others, deserves most careful inquiry and investigation. It is stated that there are at the present day more feeble-minded children of the metropolis, in proportion to population, than ever existed before, and it is computed that there is one such for every 180 healthy children who requires special educational care and training. Not only, then, is there a lowering of physique among town-bred people, but their mental condition is also affected, and some remedies should be pointed out towards the more efficient progress of our present system of civilization.

The cult of athleticism, which has become the absorbing passion of Englishmen, old and young, must be looked upon as, on the whole, a corrective of neurotic heredity, but it is not certain whether excessive exercise does not lead to overstrain of the circulatory system, for which future generations may have to pay heavy penalties. Already cardiac diseases, unknown in the

early years of the last century, are calling for special methods and institutions for their treatment and cure. The seriousness of high arterial tension is being discussed on all sides, and tonics, in the form of phosphorus, arsenic and strychnia are much more common, as also are cardiac sedatives. In Germany much attention is paid to suitable open-air playgrounds exclusively for children, and teachers are appointed to play with them. There is no doubt that physical culture is one of the greatest needs for our young people of to-day. I can speak in high praise of Swedish drill as a successful addition to the treatment of the insane. Its use has been very beneficial in the experience of my colleagues and myself at Claybury. It is a system which connects mental and muscular processes, engages the attention, and ensures a more precise and ready reaction to outward stimuli.

What have been the psychical effects of "civilization?" As regards education the present system has doubtless raised the general intelligence of the community, but it has destroyed individuality, it does not train the eye to see, the hand to work, nor the imagination to create. Few men of the first rank now rise from the masses. It is also said by those who know that respect for personal qualities has disappeared, and has given place to respect for the longest purse. This adoration of the millionaire demonstrates the craving for luxury, and the means to obtain pleasure, and it permeates every rank of society. Men now think they have a right to gratify every passion irrespective of the misery this may involve to those dependent upon them. The question is whether the pendulum has not swung dangerously far in the direction of toleration, and whether liberty has not degenerated into license. Already there are not wanting signs that legislation is being called for to deal with prodigals, spendthrifts and persons guilty of gross self-indulgence. There is no doubt that to-day altruism has lessened and individual selfishness has greatly increased. Our hospitals languish for lack of funds, charity has never been doled with less liberality, and our great universities, the pride of our country in the middle ages, make futile appeals for support. Benevolence, as in past centuries, does not in our day found great institutions for charity, education, and religious devotion. Further, the judicial statis-

tics (1903) show that certain kinds of law-breaking are also on the increase; there are more divorces than ever among the upper classes; the petitions for dissolution of marriages in 1901, the last year reported upon, were higher than in any previous year. Orders made by the Summary Court for judicial separation have increased by 31 per cent. It is only too apparent that there is less general heed paid to marital constancy, and the laxity of morals among the "smart set" of women cannot but exercise a baneful influence upon the future morals of this country. Commercial morality has declined, for crimes of embezzlement and betrayal of trust on the part of trustees, managers, secretaries, clerks and the like have shown a decided tendency to increase. There is a dearth of sincerity, and of men who "run dead straight."

As to the sharp differentiation and subdivision of workmen into groups, pointed out by Charles Booth,[1] is it now certain that these groups do not hurry to get through their tasks so as to "enjoy themselves" in some banal and senseless amusement, and that their work is performed indifferently, and without pleasure, in contrast to the individual attention from the workmen of former days, who, as stated, showed their joy in work well done? It must not be asserted that when wages are high the people are contented. The reverse is often the case, and it would be true to say that there has never been an age more discontented than the present. Socialism, that feeling of the body politic in the aggregate which levels down rather than up, and which provides for the many at the expense of the few, tends to the extinction of individual enterprise of every kind. It tolerates, if it does not sanction and encourage, the present state of nearly one-fourth of the wage-earning population who are reported to exist at rates of wages just above starvation, and who never rise with nor participate in the country's prosperity. This socialism is favored, if not determined, by the fact that the preponderating vote of the elective body decides the policy of the elected body. Under this system among other developments, a vast amount of municipal indebtedness has been incurred, and so serious is this, that the President of the Incorporated Associa-

[1] Life and Labours of the People.

tion of Municipal and County Engineers (Mr. Weaver) recently called attention to the increase of 216 per cent in local indebtedness which has taken place during the last quarter of a century, and which, to prevent municipal bankruptcy, can only result in the nationalization of municipal loans. The spirit of chivalry and courtesy, already referred to, has certainly all but disappeared. The difficulties of traveling in past days made women much more dependent upon men than they are to-day, for travelers in their hurry and scurry to "catch the Tube" have no time for courtesies and no occasion for gallantry. On the other hand, women, being more free, are more addicted to pleasure than they ever were before, and they find this in the excitement of gambling, for they have become more the companions of men in sports and pastimes. It is also certain that the women of to-day drink more alcohol than formerly, and to a great extent secretly. As to the general question of alcohol, the subject will be dealt with fully in the debates of the section, and I shall not further allude to it here.

From what has already been said (and we are much indebted to Stanley Little for his records[2]) it is seen that high pressure, and a subdivision of labor, have evolved a very complicated mental mechanism with every possible liability, therefore, of getting out of order, which has resulted in the appearance of a large variety of mental diseases unknown to the older physicians. We shall see the import of this in our comparison of the diseases of to-day with those of a hundred years ago.

It is interesting to note that the first serious legal enactments in our country respecting the care of the insane do not date back farther than the second or third year of the reign of William IV. By the courtesy of the Lunacy Commissioners, I am informed that some statistics date as far back as the reign of Edward II. Possibly the mental infirmity of King George III gave an impetus to the more enlightened modern treatment of mental diseases, which, until about this period, was not only a reproach, but also highly reprehensible. Before this reign we have no noteworthy records of asylums other than Bethlem. St. Luke's Hospital had been opened less than ten years, but from

[2] Progress of the British Empire in the 19th Century.

1762 to 1822 we have valuable contributions in regard to mental diseases from Cox[3] of Fishponds, Arnold[4] of Leicester, and Haslam[5] of Bethlem Hospital, an institution in which the skilful and kindly treatment of mental diseases was then, as now, an honor to our land. About the year 1793 we have the work of that great champion of the rights of the insane—Pinel, of the Bicêtre—translated into English by Davis,[6] of Sheffield, a philosopher, a scholar and a physician. Haslam was the first to describe general paralysis of the insane, a disease which is probably the most highly evolved form of mental breakdown, and at the opposite pole to the primary defects of idiocy and imbecility. General paralysis was then rare, but is now so common that Dr. Clouston, in his last report of Morningside, considers it to be in the proportion of 21.7 for male admissions, and 6.7 for female admissions, as against 17.3 males and 3.3 females for the whole of the London County asylums. Whether the disuse of mercury as a general therapeutic remedy in our day, compared with the frequent salivation for the smallest ailments in the past may have anything to do with this increased prevalence is uncertain and too speculative to be considered here.

There is reason to think that syphilis is more common now than a century ago (although the opinion of Sir Alfred Cooper dissents from this view), and it is possible that its dire effects upon the nervous system might be controlled or modified, if not altogether avoided, if these were more generally known, or if information respecting the far-reaching effects of syphilis and the nature of the disease were more widely disseminated. The ravages in India among our troops has given rise to loud cries for special protective legislation against contagious diseases of a luetic character, and only last year a unanimous resolution was conveyed from the Section of Psychological Medicine of this Association urging the attention of the Government and

[3] Cox. "Practical Observations on Insanity," 2nd Ed., 8vo, London, 1806.

[4] Arnold. "On the Management of the Insane," 8vo, London, 1809.

[5] "Observations on Madness and Melancholy," by John Haslam, of Pembroke Hall, Cambridge, Apothecary to Bethlem Hospital, 8vo, London, 1809.

[6] Pinel's "Treatise on Insanity," trans. by Dr. Davis, 8vo, Sheffield, 1806.

public bodies to the horrible results of this disease. But our statesmen are timid about the health of the people when fanatics, faddists, conscientious objectors and other irresponsible agitators begin to make political capital out of the "moral" question. It is not too much to say that the more highly developed a race becomes the more cases of general paralysis and other forms of insanity due to the same cause will occur, and it is not too much to infer that the diseases named are preventible and should be prevented.

Haslam refers, before the year 1800, to the disease described as *dementia præcox,* then so rare, but now so common that it may almost be described as the scourge of our asylums, for it attacks prematurely our most promising youth, it is practically incurable, and will help to fill our asylums of the future with the hopelessly insane. It is, of all forms, the one caused by overstrain, and the mental worker rather than the manual is subject to its ravages. Moreover, as has been stated, the great majority never recover from it. This is Haslam's description of it in 1800: "A species of insanity, sometimes occurring about the time of puberty, especially in those who have possessed a good capacity and a lively disposition, and in females more frequently than males; their faculties are gradually obliterated until they are at last complete and incurable idiots." That which was then occasional may now be described as having become a common variety of insanity among our promising and educated youth.

Haslam reported, prior to 1806, upon the treatment of 8874 cases of insanity (4042 males, 4832 females) in the second Bethlem Hospital, built in Moorfields in 1676, and the result of 46 years' experience. The maniacal form of insanity was then more common. Those were the days when men were content and the nation had faith in its destiny. In the *Times* of July 11, 1803, was published a war-song, which, after inviting the Consul-King to attack us, proceeds:

> . . . . . that Britons
> Can fearless still maintain their stand,
> And single-handed crush the pride of France.

All classes then bore the necessary hardship of high taxation with resignation and without discontent.

As to the recovery rate, Haslam states that his own, among cases of mania, was 67 per cent. Last year, in all the asylums of London, the recovery rate from all cases of mania amounted to 38.4 per cent. We had few insane in those days, but those which occurred were acute and curable. Not until January 1, 1859, was the number of lunatics officially registered in this country; at that date there were 36,762 insane persons—a proportion to the population of 1 to 536. To-day they number over 113,964, a proportion to the population of 1 to every 293, and the tendency of much of the insanity which comes under treatment to-day is to end in dementia and to become incurable. Even a quarter of a century ago the type of insanity was different from that of to-day. There is an increased tendency to melancholia, which is less recoverable than mania, being probably a deeper reduction of nerve elements than occurs in mania. Melancholia has shown a considerable rise among the educated and private class of the insane, and I am stating a fact, and not an opinion, when I say that recovery may be complete after a sharp attack of mania, whereas this is rarely the case after melancholia, more especially in men. This supports the opinion of the earlier physicians that "furious insanity is much more curable than melancholy madness," both Haslam and Pinel having recorded this observation.

Of all the 9267 patients (4096 males, 5171 females) who have been under treatment in the Claybury Asylum since its opening, a period of ten years, and of whom I have direct personal experience and record, 1132 males and 1625 females recovered, a percentage of 27.2 males and 31.5 females. Of the 8874 at Bethlem Hospital (4042 males, 4832 females) referred to above, and under treatment over 100 years ago, 1155 men and 1402 women recovered, a proportion of 27 per cent for the men and 29 per cent for the women, an improvement in the recovery rate of to-day of just over 1 per cent, in spite of all the means of treatment which a century's progress in applied art and the sciences has placed at our disposal. Surely there must be some other explanation than the failure of modern therapeutic means! May not this be discovered in the change type already referred to and incident to our civilization?

If a comparison is made between the reports of the Lunacy

Commissioners for the years 1878 and 1902, a period of 25 years, which, as stated, is within my experience, a reduction is seen in the percentages of all cases of acute insanity recorded, a decrease from 75.3 to 70.9 per cent, and further, as cases of *dementia præcox,* or premature dementia in young persons, already referred to, were not recorded in the statistics of 1878, and they almost invariably commence in depression, it is justifiable to conclude that they were classed as melancholia, which has risen from 21.5 per cent in 1878 to 27.9 per cent in 1902, the reduction in cases of mania in these years being from 53.8 per cent to 43 per cent. The age periods in the statistics of these Blue Book reports are not given, but the calculations ascertained are absolutely confirmatory of the statements I have made. As to the recovery rate, the Blue Book for 1878 gives it, for males as 34.81 per cent, and for females 42.84 per cent, an average total of 38.84 per cent, whereas for the year 1902 the recovery rate is given as 34.4 per cent for males, 39.7 per cent for females, an average total of 37.1 per cent, and actual decrease in the recovery rate for the first and last of the 25 years under review.

With the progress of civilization, therefore, not only is insanity on the increase, but the occurring varieties are less curable; the physique of the town-dwelling section of the population has deteriorated both in height and weight, and the statistics of recovery are less favorable to-day than they were 25 years ago. Serious as this may appear, while such a state exists, one can neither hope for nor expect relief from the great financial and economic burden of providing accommodation for the insane in the future. If relief is to come, it will be in some great change affecting the physical as well as the mental health of the masses of the people, a foreshadowing of which is beyond the scope of this paper.

There is yet one factor in the physical condition of the people which affords opportunities for high ideals, which kindles a spirit of sympathy, devotion and fervor even among the roughest characters, and in the most crowded areas of our cities. I refer to the effect of spiritual influence. In an assembly such as ours, I touch upon it with extreme diffidence and humility, but it is an element in human character for which even the Prime Minister has recently appealed, and the life of Father

Dolling, the work of such institutions as the Salvation and the Church Armies, testify to its influence over the mental and moral character of those hopeless persons who become the deposit of our civilization. The indifference of the great bulk of the people to this aspect of life is a matter of common knowledge, and an interesting contrast has been recently furnished in the July number of the *Edinburgh Review*. At the present time there are, in London alone, over 700,000 young men between the ages of 16 and 21—and mostly sons of working men —who need special guidance and training. It is not improbable that "Hooliganism" and other forms of social outbursts might be much modified through an influence from this direction.

Everything points to the strong and immediate necessity for preventing the extension of this lowered mental and physical vigor. Our zeal, energy and skill should be directed to assist a more healthy adjustment of the mind of man to his ever-varying and progressively complicated environment.

THE BLOOD IN EPILEPSY: EXPERIMENTS ON ANIMALS.[1]

(PRELIMINARY REPORT.)

BY F. SAVARY PEARCE, M. D.,

Prof. of Nervous and Mental Diseases in the Medico-Chirurgical College of Philadelphia,

AND

L. NAPOLEON BOSTON, A. M., M. D.

Bacteriologist to the Philadelphia Hospital, Demonstrator in charge of Clinical Laboratory, Medico-Chirurgical College.

The difficulties of the study of diseases lie chiefly in the determination of pathogenesis and etiology. In many nervous affections the causes are legion. What may be the primary or initial exciting factor in the production of one of the most enigmatic of all diseases—the so-called idiopathic epilepsy—has never been made out. The object of this experimental study is to determine if changes in the blood may be a possible cause, in part at least, of this terrible disease, in which there must exist unstable neuron function and irritability of the motor cortex.

We have excluded every case of epilepsy in the experimentation where there was known organic disease resident in the brain or its membranes; in the kidneys, heart, or other organs; and have, therefore, limited the cases studied to those of essential epilepsy as we understand it to-day in the light of the meagre scientific data upon this subject. The cases were all selected with great care as to the above outline from the nervous wards of the Philadelphia Hospital. Dr. S. R. Seifert assisted us in the blood counts made upon the patients and the animals. We desire to thank this gentleman for his aid in the work.

The plan of such a study must be broad, lest false deductions

[1] Read April 1, 1903, before the College of Physicians of Philadelphia.

be easily made. Appreciating such a desideratum, the writers in this necessarily preliminary contribution are well aware of its limitations. We hope to pursue the study further as new light may present to guide in the future. Attempt has been made here, as indicated, to exclude organic cases of epilepsy, such as those following trauma, fracture of the skull, meningitis; also congenital cases, believing that such intrinsic known causes for the then symptomatic attacks are explained by direct irritation to the motor cortex, epileptic seizures following.

But what causes the more subtle attacks designated to-day as essential is the more difficult problem. We do know that complicating diseases, as the acute infectious disorders, will frequently stay "attacks" for more or less long periods of time; that pregnancy will do the same thing in some cases, but you *never* know in which case. Theories come fast enough as to the *why* of it all. Can antitoxins or incompatible poisons of other diseases counteract a poison which is occasionally accumulating and ordinarily exciting attacks, etc.? Can a sudden fright produce such a restored competence of the chemistry of the body in rare cases that no such toxins are further developed and the epilepsy is cured? Dr. Weir Mitchell has told us (Pearce) of a case where the excitement of battle in the period of our Civil War seemed to be *the* curative agent.

Since no definite histological change has been found in acquired idiopathic epilepsy, inherent irritability of the cortical cells remains as an only explanation for the existence of the disease, if we do not think of disturbed metabolism being, perhaps, the exciting cause, or of some other poison circulating in the blood. We know very well clinically that constipation will precipitate attacks in most patients the subjects of epilepsy. It is not definitely known whether this is a simple reflex or due to absorption of toxins.

Hence it is that blood studies are most desirable in epileptics, as is also the careful observation of the urine and other bodily excretions and secretions. But as the blood-vessels ultimately must carry any abnormal substance to the brain to act as exciting causes of attacks, the poison *must* at times be resident, therefore, in the blood serum or in the corpuscles.

For this reason we made observations on *seven* cases of idiopathic epilepsy as to the number of red and white blood cells

present per cmm.; the percentage of hæmoglobin; differential leukocyte counts in the patients before and after epileptic seizures; and in animals, both before and after inoculation with defibrinated blood of epileptics, with a view of ascertaining the degree of toxicity common to epileptic bloods. The accompanying experiments will elucidate our findings:

Blood-Count Series of Seven Cases of Idiopathic Epilepsy.

CASE I.—June 16. E. McL., aged sixteen years; subject to convulsions for the last ten years about twice a week, becoming dizzy, then falling; never bit tongue nor sustained personal injury in falling. Does not void urine during attacks. His general appearance is good; heart, lungs, and reflexes appear normal. Hæmoglobin, 74 per cent; red blood corpuscles, 5,190,000 white blood corpuscles, 10,600. Differential leukocyte count: polymorphonuclears, 68 per cent; large lymphocytes, 2 per cent; small lymphocytes, 18 per cent; large mononuclears, 5 per cent; eosinophiles, 0.5 per cent; broken eosinophiles, 0.5 per cent; myelocytes, 3.5 per cent; transitionals, 2.5 per cent.

CASE II.—July 28, 1902. H. R., aged twenty years. Family presents no epileptic history; has had attacks since six years of age; occasionally has spells of melancholia, never has an aura; attacks most frequent at night, irregular in time. General appearance good; general condition good. Hæmoglobin 73 per cent; red blood corpuscles, 5,490,000; white blood corpuscles, 18,000. Differential leukocyte count: polymorphonuclears, 72 per cent; large lymphocytes, 4 per cent; small lymphocytes, 15 per cent; large mononuclears, 9 per cent.

CASE III.—W. H., aged thirty-five years; has been subject to convulsions all his life; major convulsion every two or three weeks, with an occasional interval of four weeks. General condition good; heart and lungs normal. Hæmoglobin, 85 per cent; red blood corpuscles, 5,520,000; white blood corpuscles, 13,000. Differential leukocyte count: polymorphonuclears, 62.5 per cent; large lymphocytes, 2.5 per cent; small lymphocytes, 20. 5 per cent; eosinophiles, 0.5 per cent; broken eosinophiles, 2 per cent; transitionals, 3.5 per cent; large mononuclears, 5 per cent; polymorphonuclears and eosin granules, 0.5 per cent.

CASE IV.—July 31, 1902. J. F., aged forty-seven years, has been subject to convulsions at irregular intervals since his eighth year, at present about seven per month. General appearance good; general condition good. Blood count: hæmoglobin, 76 per cent; red blood corpuscles 4,610,000; white blood corpuscles, 7400. Differential leukocyte count: polymorphonuclears, 29 per cent; large lymphocytes, 17 per cent; small lymphocytes, 13 per cent; eosinophiles, 1 per cent; broken eosinophiles, 1 per cent; large mononuclears, 37 per cent; transitional, 2 per cent.

There were in this case also a few megalocytes, a few shadow cells, a few poikilocytes, and several cells that appeared to be broken polymorphonuclear cells.

CASE V.—August 6, 1902. L. F., aged fifty-three years, has had convulsions every week or two for the last fifteen years, occurring sometimes once a month. General appearance and physical condition is good. Blood count: hæmoglobin, 86 per cent; red blood corpuscles, 5,060,000; white blood corpuscles, 11,500. Differential leukocyte count: polymorphonuclears, 42.5 per cent; large lymphocytes, 12.5 per cent; small lymphocytes, 16.5 per cent; large lymphocytes, 4 per cent; eosinophiles, 1 per cent; broken eosinophiles, 2 per cent; large mononuclears, 19 per cent; transitionals, 4.5 per cent; myelocytes, 2 per cent.

CASE VI.—August 8, 1902. J. Y., aged thirteen years, has had convulsions of irregular occurrence for about the last five years, getting dizzy and falling, froths at the mouth, does not bite the tongue. General appearance good; general condition good. Blood count; hæmoglobin, 63 per cent; red blood corpuscles, 5,620,000; white blood corpuscles 11,400. Differential leukocyte count: polymorphonuclears 43.5 per cent: large lymphocytes, 3 per cent; small lymphocytes, 30 per cent; eosinophiles, 3 per cent; large mononuclears, 15 per cent; myelocytes, 2.5 per cent; transitionals, 3 per cent.

CASE VII.—P. C., aged fifty-eight years, has had convulsions since birth; very nervous, bites the tongue, grits the teeth; has convulsions about four to six times per month, irregular in time. General physical condition good; general appearance fair. Blood count: hæmoglobin, 76 per cent; red blood corpuscles, 4,890,000; white blood corpuscles, 21,200. Differential leukocyte count: polymorphonuclears, 69.5 per cent; large lymphocytes, 3.5 per

cent; small lymphocytes, 14.5 per cent; eosinophiles, 3.5 per cent; broken eosinophiles, 1 per cent; large mononuclears, 9.5 per cent; transitionals, 3.5 per cent. Slight poikilocytosis, and a moderate pallor of red cells.

CONCLUSIONS.—From these seven blood estimations from as many cases it would seem that there is in idiopathic epilepsy a fairly well-marked chlorotic type of anæmia. There was a leukocytosis, too, in all but one of the seven cases where the estimation showed 7400 white cells per cmm. In one the count was 18,000; in another 10,000; in another 13,000 cells per cmm. These counts were made three to four hours after a limited meal in all cases.

The findings by differential leukocyte counts cannot be said to be of striking interest; yet worthy of mention are the facts that the polymorphonuclear cells were reduced in three instances equally in 29, 42, and 43 per cent, respectively. This decided reduction in the polynuclear cells was not associated with an increase in the lymphocytes, as is the case where this change be induced by malnutrition, starvation, etc. Myelocytes were found in three instances reaching a maximum of 3.5 per cent. The red cells were approximately normal in numbers. In all the bloods there was a moderate degree of poikilocytosis, and this finding was especially marked in those where the hæmoglobin was greatly reduced, the degree of distortion bearing a direct correlation to the loss in hæmoglobin.

There were to be seen here and there red cells showing unstained punctate areas scattered throughout their protoplasm. Punctate or granular basophilia, apparently identical with that seen in lead-workers, was present in the blood from three of these cases.

The above examinations, therefore, revealed no conclusive evidence of change *per se* in the cells themselves in the epileptic. But it still is possible that a toxin may be circulating, being held in suspension in the blood serum, and which may excite attacks.

Apropos of this, at a time four or five years ago, from a case of idiopathic epilepsy, one of us injected 20 cc. of defibrinated blood into the vein of a case of pernicious anæmia. About seven days later the case had two epileptic seizures, and was followed by one or more attacks daily for a period of four or five days,

when he died from the exhaustion of the anæmia. Dr. F. P. Henry has performed a similar inoculation in the presence of one of us (Boston), and here, too, the case of anæmia developed true epilepsy.

As control test the blood of two healthy students was studied, as was that of epileptics. The blood from each student was injected into the peritoneal cavities of two healthy rabbits, and the blood of these animals examined daily for one week, finding no alteration whatever in the hæmoglobin, red cells or leukocytes.

We also took typical cases of idiopathic epilepsy with the intent to make experimental study on healthy rabbits, counting the rabbits' blood before and after the injection of defibrinated blood antiseptically drawn from the epileptic patient. We found blood changes in rabbits injected with the blood of idiopathic epilepsy (Cases VIII and IX) given below.

We will leave the experiments now to be recited to speak for themselves, believing that suggestions from this work will be manifest, and that, presumably, some hæmolytic enzyme is a part, at least, of the pathogenesis of essential epilepsy.

In order to obtain an average blood count for the healthy rabbit when kept in captivity, and subjected to the environments characteristic of the laboratory, we found the hæmoglobin between 65 and 75 per cent, 68 to 70 being the percentage for most animals; yet at times animals apparently well nourished showed blood which gave a much lower reading. Examination of the average number of erythrocytes in twenty animals, examined at the laboratory, showed these cells to vary in numbers between 4,500,000 and 5,100,000 per cmm. The leukocyte counts taken from the same records were found most often between 5000 and 6000; and in but a few instances did these cells exceed 8000 per cmm. Differential leukocyte counts were far less constant, and, in fact, were seen to vary greatly in the same animal at different days, independent of inoculation.

Blood counts of two idiopathic cases of epilepsy were made on June 7, 1902. Case VIII was experimented with as to non-defibrinated and defibrinated blood injected into rabbits (see below).

Case VIII.—The first case, W. H., male, aged thirty-five years; blood examined after last of four severe seizures in twenty-four hours, which were on June 1, 1902. Blood count normal;

exact count mislaid. The differential leukocyte count was: polymorphonuclears, 78 per cent; large lymphocytes, 6 per cent; small lymphocytes, 7 per cent; myelocytes, 7 per cent; transitional myelocytes, 2 per cent.

June 13, 1902. W. H. was bled to-day. He has had no attack in the interval. The blood was drawn by hypodermic syringe from the median cephalic vein, and 10 m. were injected subcutaneously into a small rabbit. The count of this rabbit before injection was normal.

Results in rabbit from above two injections: June 17th. Four days after first injection, and twenty-four hours after second. Rabbit (A)—small rabbit: hæmoglobin, 46 per cent; red blood corpuscles, 4,490,000; white blood corpuscles, 8000. Differential leukocyte count: polymorphonuclears, 35 per cent; large lymphocytes, 21 per cent. (*i e.*, cells which corresponded to the large lymphocytes in man).

Small lymphocytes, 10.5 per cent; eosinophiles (very irregular as to size of granules) 29 per cent; myelocytes, 3.5 per cent; normoblasts, 1 per cent; poikilocytosis marked.

June 19, 1902. W. H. No attacks since June 7th. Bled again to-day, and 20 m. injected for the third time into a rabbit. Rabbit showed marked trembling shortly after inoculation, lasting about half a minute, but no true convulsion.

June 20th. Count of small rabbit (A) seven days after first injection, four days after second, and twenty-four hours after the third: hæmoglobin, 55 per cent; red blood corpuscles, 4,680,000; white blood corpuscles, 5600. Differential leukocyte count: polymorphonuclears, 15 per cent, all showing some eosinophilic granules; large lymphocytes, 14 per cent; small lymphocytes, 25 per cent; eosinophiles, 40 per cent; myelocytes, 6 per cent.

June 25th. Count of gray rabbit (B), W. H's *non-defibrinated* blood, six days after inoculation: hæmoglobin, 48 per cent; red blood corpuscles, 3,960,000; white blood corpuscles, 7200. Differential leukocyte count: polymorphonuclears, 18 per cent; large lymphocytes, 24 per cent; small lymphocytes, 18 per cent; eosinophiles, 2 per cent; polymorphonuclears showing eosinophile granules, 36 per cent; myelocytes, 2 per cent.

Normal count of rabbit (B) before defibrinated blood injection experiments.

June 26th. Count of gray rabbit (B) before inoculation with defibrinated blood of W. H.

Differential count: polymorphonuclears, 4 per cent; polymorphonuclears with fine eosinophile granules, 16 per cent; large lymphocytes, 35 per cent; small lymphocytes, 15 per cent; myelocytes, 5 per cent; myelocytes with eosinophile granules, 1 per cent; eosinophiles, 24 per cent.

Defibrinated Blood Experiments.

June 26th. W. H. (Case VIII, same as Case III); no convulsions. Bled this afternoon and 45 m. of defibrinated blood injected into peritoneal cavity of small gray rabbit (C).

June 27th. Twenty-four hours after rabbit was injected with 45 m. of defibrinated blood from W. H.: hæmoglobin, 79 per cent; red blood corpuscles, 6,870,000; white blood corpuscles, 8600. Differential leukocyte count: polymorphonuclears, 23 per cent; polymorphonuclears with eosinophile granules, 55 per cent; large lymphocytes, 13 per cent; small lymphocytes, 9 per cent; eosinophiles, 9 per cent.

June 30th. Patient had no attacks; bled, and 45 m. of defibrinated blood injected into gray rabbit (B); peritoneal cavity.

June 30th, same rabbit. Hæmoglobin, 84 per cent, a slight rise (5 per cent); red blood corpuscles, 6,980,000; white blood corpuscles, 12,400 (a rise of 3800 cells). Differential count: polymorphonuclears, 8 per cent; polymorphonuclears with fine eosinophile granules, 41 per cent; large lymphocytes, 8.5 per cent; large lymphocytes with eosinophile granules, 1 per cent; small lymphocytes, 28.5 per cent; eosinophiles, 11.5 per cent; large mononuclears, 1.5 per cent. After above count 45 m. of defibrinated blood injected into peritoneal cavity of the rabbit (B).

July 1st. Twenty-four hours after injection, as above, rabbit showed some indisposition to eat. Lay crouched in corner. Hæmoglobin, 77 per cent, a slight fall (7 per cent); red blood corpuscles, 6,370,000, no marked change; white blood corpuscles, 17,000, a rise of 4600 cells. Differential leukocyte count: polymorphonuclears, 4 per cent; polymorphonuclears with fine eosinophile granules, 25 per cent; large lymphocytes, 9 per cent; small lymphocytes, 41 per cent; large mononuclears, 9 per cent; eosinophiles, 8 per cent; transitional leukocytes, 2 per cent; myelocytes, 2 per cent; moderate poikilocytosis marked.

July 2d. Forty-eight hours after above injection: hæmoglobin, 78 per cent; red blood corpuscles, 6,610,000; white blood corpuscles, 17,500. Differential leukocyte count: polymorphonuclears with eosinophile granules, 29 per cent; large lymphocytes, 4 per cent, a fall; small lymphocytes, 9 per cent, *fall of 37 per cent in twenty-four hours*; large mononuclears, 3 per cent; eosinophiles, 54 per cent, a rise; transitional leukocytes, 1 per cent; moderate poikilocytosis, few shadow cells.

July 3d. Seventy-two hours after injection this rabbit showed indisposition to move about; also coughed some and had some mucous discharge from nose. Hæmoglobin, 44 per cent, a loss of 35 per cent from normal; red blood corpuscles, 6,570,000; white blood corpuscles, 16,000. Differential count: polymorphonuclears with fine eosinophile granules, 51 per cent; small lymphocytes, 15 per cent; large lymphocytes, 3 per cent; eosinophiles, 2 per cent; eosinophiles irregular and broken, 13 per cent; large mononuclears, 12 per cent; large mononuclears with eosinophile granules (?), 1 per cent; transitionals, 1 per cent; transitionals and eosinophile granules, 2 per cent.

July 5th. W. H. has had no attacks, but complains of general weakness and shows questionable physical signs of phthisis. Was bled again, and 60 m. of defibrinated blood injected into gray rabbit(B), peritoneal cavity.

July 5th. Rabbit to-day injected with 60m. of defibrinated blood from W. H.

July 6th. Rabbit showed tremors and rigidity over entire body for about one hour this morning; still coughs some, and at times sonorous respiration. Differential count: polymorphonuclears, 1.5 per cent; polymorphonuclears with fine eosinophile granules, 30 per cent; large lymphocytes, 5.5 per cent; small lymphocytes, 37 per cent; large mononuclears, 5 per cent; eosinophiles, 9 per cent; broken eosinophiles, 12 per cent.

July 8th. W. H. has had no attacks. Complains of continuous cough. Anorexia. Very weak.

July 9th. Sputum examination for tubercle bacilli negative.

July 10th. Five days after last injection of rabbit: white blood corpuscles, 10,600. Differential count: polymorphonuclears, 3 per cent; polymorphonuclears with fine eosinophile granules, 26 per cent; large lymphocytes, 14 per cent, an increase; small lym-

phocytes, 20 per cent; eosinophiles, 5 per cent; eosinophiles broken and irregular, 11 per cent; myelocytes, 10 per cent; large mononuclears, 9 per cent; transitionals, 2 per cent.

July 11th. W. H. has had no attacks. Bled, and 80 m. of defibrinated blood injected into gray rabbit (B), peritoneal cavity.

July 12th. No attacks; 90 m. injected into peritoneal cavity of gray rabbit (B).

July 14th. Gray rabbit (B) inoculated with 90 m (*twice the usual dose*) blood of W. H. into peritoneal cavity. Rabbit showed marked tremors of both forelegs all day; also appeared stupid. Blood count twenty-four hours later: white blood corpuscles, 162,800—*enormous rise in leukocytes.* Differential leukocyte count: polymorphonuclears, 2.5 per cent; polymorphonuclears with fine eosinophile granules, 38 per cent; large leukocytes, 10.5 per cent; small leukocytes, 21.5 per cent; large mononuclears, 8.5 per cent; eosinophiles, 3.5 per cent; eosinophiles broken, 8.5 per cent; myelocytes, 3 per cent; transitional leukocytes, 5 per cent; marked poikilocytosis.

July 17th. Count of gray rabbit (B) three days after last injection: leukocytes, 11,000, a decided drop, but still above normal. Differential leukocyte count: polymorphonuclears, 2 per cent; polymorphonuclears with fine eosinophile granules, 53 per cent, a rise toward normal; large lymphocytes, 6 per cent; small lymphocytes, 15 per cent; eosinophiles, 9 per cent; broken eosinophiles, 7 per cent; large mononuclears, 4 per cent; myelocytes, 1 per cent; transitionals, 1 per cent.

An analysis of the results of these two sets of careful experiments with the blood of epilepsy (*one with blood as it was drawn from the patient, and again where it was defibrinated*) were about the same.

The defibrinated blood results can be stated in one case as follows: It will thus be seen that the hæmoglobin rose from 65 per cent to 79 per cent and later to 84 per cent; then, finally, to 44 per cent, seven days after last injection. The leukocytes in twenty-four hours rose from 8000 to 12,000 after first injection; and after second injection from 12,000 to 17,000. In both of these injections 45 m. of defibrinated blood was used. On July 11, 1902, 80 m. more of defibrinated blood was injected intto the peritoneal cavity of the same rabbit; on July 14th the injection

was again repeated, giving 90 m. of defibrinated blood (about twice the dose usually given). All day long the rabbit was very ill (was rigid and tremulous), and twenty-four hours later its white blood cells reached the enormous number of 162,800 per cmm., but then rapidly fell to 11,800 two days later. This must show some extreme toxic condition of the blood in the epileptic man at this time, from whom the blood was taken. It was capable of producing a profound leukocytosis, which very soon disappeared in the rabbit.

The eosinophiles fell after injection of the rabbit from 24 per cent to 9 per cent, but on the sixth day after first large injection (twenty-four hours after last large dose) rose again to 54 per cent. Eighteen days after injection, when the leukocytes were highest, too, the animal showed that he was very ill, with especial tremor of the front legs, etc. At this date the eosinophiles were 13 per cent (about normal). The polynuclears rose from 18 per cent to 36 per cent twenty-four hours after injection, with marked reduction in the mononuclear cells; also when the large mononuclear cells went up the lymphocytes went down, and *vice versa*. The red blood cells also showed marked changes, poikilocytosis, etc.

CONCLUSIONS:—It would seem from the experiments in this case (VIII) that there was a profound toxæmia set up in the rabbit, as shown by the definite blood changes seen in the animal. There was no corpuscular change noted in the patient; therefore, if the blood changes in the rabbit were due to alteration in the blood it was principally in the serum—a toxic process. That the patient's cells were not disturbed in the differential count does not imply that such marked changes as we find in this rabbit are not due to a toxin of some sort from his blood, whose own cells may have become immune.

CASE IX.—J. D., male. Second case bled June 8, 1903. Blood count after five attacks in five days was mislaid. Hæmoglobin, 63 per cent. Differential count in this patient: polymorphonuclear cells, 70 per cent; large lymphocytes, 13 per cent; small lymphocytes, 11 per cent; eosinophiles, 1 per cent.; myelocytes, 4 per cent; transitionals, 1 per cent. There was present a degree of moderate poikilocytosis, and many of the red cells were pale and showed punctate areas of pallor.

June 13, 1902. J. D.; since bled has had following attacks: June 9th, 4; June 10th, 2; June 11th, 3; June 12th, 1; June 13th, 4, up to 5 p. m., at which time he appeared quite stupid and averse to answering questions. His knee reflexes were much reduced. Following an attack blood was drawn, and 20 minims were injected intraperitoneally into a large rabbit.

June 16th. J. D., very stupid for last two weeks, has had following attacks: June 14th, 6; June 16th, 2. Following a slight attack blood was drawn, and 20 minims were injected intraperitoneally into this animal.

Normal blood count of rabbit (C): Hæmoglobin, 50 per cent; red blood corpuscle count lost, but was about normal; white blood corpuscles, 9000.

June 17th. The large rabbit four days after first and twenty-four hours after second injection showed: hæmoglobin, 50 per cent; leukocytes, 14,200; red cells, 6,180,000. Differential leukocyte count: polymorphonuclears, 39.5 per cent; small lymphocytes, 41.5 per cent; large lymphocytes, 10 per cent; eosinophiles, 2 per cent; myelocytes, 7 per cent. Many normoblasts were present.

June 19th. J. D. bled this afternoon at three o'clock, and 20 minims defibrinated blood injected intraperitoneally into the same animal.

June 20th. Twenty-four hours after last injection. Hæmoglobin, 63 per cent; red blood corpuscles, 5,890,000; white blood corpuscles, 10,500. Differential count: polymorphonuclears, 6 per cent; large lymphocytes, 24 per cent; small lymphocytes, 8 per cent; eosinophiles (irregular), 56 per cent; myelocytes, (?) 6 per cent; moderate poikilocytosis.

June 20th. J. D. died at 4 p. m. during a convulsion. Rabbit count to be continued without further injection.

June 25th. Large rabbit, six days after third injection, showed: hæmoglobin, 66 per cent; red cells, 4,940,000; leukocytes, 18,800. Differential leukocyte count: polymorphonuclears, containing numerous eosinophile granules, 15 per cent; polymorphonuclear (true), 3 per cent; large lymphocytes, 23 per cent; small lymphocytes, 8 per cent; eosinophiles, 46 per cent; myelocytes, (?) 5 per cent. There was a moderate grade of poikilocytosis.

July 1st. Last count, twelve days after last injection: hæmoglobin, 64 per cent; red cells, 2,850,000; leukocytes, 10,800.

CONCLUSIONS (Case IX blood used).—Hæmoglobin in rabbit before any inoculation, about 50 per cent. Gradual rise after each injection to 66 per cent six days after last injection. Leukocytes 9000 before injection; 14,000 twenty-four hours after the first injection, and fluctuated between 10,000 and 18,000 after each successive injection until the sixth day after last injection. Red blood corpuscles fell from 6,000,000 to 2,850,000 twelve days after the first injection. During the period of injections the red blood cells fell over 1,000,000 per cmm., showing a progressive destructive change. The eosinophiles rose from 1 to 56 per cent after the second injection. The polynuclear cells fell from 37 per cent to 3 per cent within twenty-four hours after the last injection, and rose to 18 per cent six days later. The tendency to widespread alteration in the proportional relation of the blood cells and hæmoglobin, as well as in this rabbit; the great increase in the number of leukocytes, all coming on soon after the injection of blood from a second case of idiopathic epilepsy, point again toward toxæmia as an existent factor in this disease. We did not produce distinct convulsions in the rabbit, it is true; but the blood changes were suggestive of toxæmia resulting from the injection of defibrinated blood taken from an epileptic.

It is interesting that these changes were in striking contrast to those changes noted in the leukocytes in ordinary septic processes, where the polynuclear cells show a decided increase. After each inoculation eosinophilia developed within twenty-four hours, which was equal to that observed in the blood of persons suffering from such parasitic diseases as trichinosis and filariasis. This would appear to suggest the necessity for more careful observations as to a possible relation between epilepsy and parasitic infections.

Incidentally it should be mentioned that Bra found in 70 out of 100 cases the micro-organism *microbe* he describes, and rabbits inoculated with his organism died in clonic convulsions. The number of the "neurococci," as he calls them, increased with an impending seizure and diminished again afterward, until none could be found during the intermission.

WAS HE A PARANOIAC?[1]

By C. A. DREW, M. D.,

Medical Director, Massachusetts State Asylum for Insane Criminals.

H. B., a Hebrew, thirty-five ears old, born in England, was admitted to the State Asylum for Insane Criminals January 22, 1902, from the State Prison in Charlestown, where he had been serving a five to nine years' sentence from March 26, 1897, having been convicted as a "common and notorious thief." The medical certificate on which his commitment was based reads as follows:

CHARLESTOWN, JAN. 17, 1902.

His Excellency W. Murray Crane, etc.

We have examined H. J. B., 35 years of age, committed March 26, 1897, as a common and notorious thief, sentence of from five to nine years, and would respectfully report.

When B. was first committed he worked in the harness-shop, he began to agitate the men and was placed in Cherry Hill, where he began to write to all the authorities and to lawyers telling of the illegal actions of the officers of the prison and wishing to begin actions in law against some of them. He claims to have been admitted to the bar in Dakota. He was arrested in London, Brussels, and New York as well as Boston. Perhaps there is no inmate of the prison who has given more trouble to all concerned than he.

At our first examination he began telling in legal terms how unjustly he had been treated before his trial and claimed that from the first he was a victim of persecution and conspiracy; that his sins were sins of omission (neglecting to say his prayers often) only, and not of commission. He related how many people would be defendants in cases he would bring against his persecutors. He also showed some wood and iron that had been placed in his food. The examination lasted over two hours.

At the second examination he recognized us and began to say that we were engaging in a conspiracy to pronounce him insane in order that his enemies might get possession of his property. He rehearsed nearly all that he said at the former visit and taking his oath that he was an innocent man. That in all of the former arrests in London, Brussels, New York and elsewhere he had been exonerated, and that the so-called obtaining money under false pretences was proven to be legitimate business. He asked us to examine two bottles of medicine that had been given to him from the

[1] Read before the 59th Annual Meeting of the American Medico-Psychological Association, at Washington, D. C., May 12-15, 1903.

prison dispensary and then demanded that we should make a thorough analysis of them both, but at once claimed that we should use neither what he showed us at the former visit, nor the request for examination of the medicine at this visit as evidence of delusions on his part. He quoted law to prove that if we did not at once take steps to inform the proper authorities that he was unjustly held, improperly treated and a victim of an outrageous conspiracy, we were guilty of concealing evidence and would be defendants in suits which he would bring against us. At times he would stop his conversation and examine if any people were listening to him; at another time he would get up from his chair, go to the door, and ask for the deputy just to see if he were trying to overhear our talk, although we had told him that the deputy had returned to the yard. He is very suspicious of almost everybody that comes in contact with him. We were with him over four hours at the second examination, although we were convinced at our first that B. is a litigious paranoiac. The little legal knowledge that he has is the nucleus about which are formed all his ideas of his relations to his fellowmen and their conduct to him. He has employed no less than six lawyers since he has been an inmate of the prison and has discharged them all because he became convinced that each one had been influenced by the warden, and he has told about others he had employed and discharged.

We feel that his proper place is at the asylum for insane criminals.

It may be remarked that this certificate is long and strong. It seems to establish delusions of persecution,—one of the most characteristic symptoms of that form of mental alienation called paranoia. We learn that this prisoner has been under observation nearly five years, that he began to agitate other prisoners and was placed in "Cherry Hill" soon after his sentence in 1897. It may be explained that "Cherry Hill" is a building with large, well lighted and well ventilated rooms for the isolation of incorrigible prisoners. We learn further that the prisoner was very insistent in his efforts to bring suits at law against the prison officials and others, and from other sources we learn that in the opinion of certain friends he had a "mania" for bringing suits at law even before his last arrest and sentence. Those conversant with psychiatric literature will recall that under the names of "querulent insanity," "quarreling insanity," or "litigious insanity," the majority of Continental writers, as well as prominent alienists of England and America, have written concerning these people with a so-called mania for quarreling. The majority of medical writers have considered certain of these people insane, even when no clearly defined delusions could be demonstrated,

and have generally classed them as paranoiacs,[1] although Krafft-Ebing seems inclined to put them under the heading of "Moral Insanity," and Berkley considers the litigationists better classed with the ethical imbeciles. So far then as this propensity for litigation has weight—and we shall wish to refer to this phase of the question again—it substantiates the diagnosis of the committing physicians, for it is counted competent supporting evidence of mental degeneration by the majority, at least, of recognized authorities in mental medicine. The extreme suspiciousness of the subject is another strong pillar supporting the diagnosis, because extreme suspiciousness is a common and prominent characteristic of most all paranoiacs.

Passing the leading symptoms in review, we have a strongly litigious propensity and apparent delusions of persecution, conspiracy and poisoning, with extreme suspiciousness,—the common accompaniment of such delusions,—truly a strong syndrome on which to base a diagnosis of litigious paranoia. If the suspicions of poisoning and ideas of persecution could be established beyond peradventure as delusions, in the medical sense, and if it could be shown that the man had at some time been frank, truthful and honest, insanity would be established beyond a reasonable doubt, although we might then have doubt as to the form of insanity. But the whole evidence tends to show that the man was never truthful, frank or honest, and it is almost self-evident that a man's natural temperament, character and education, must be considered before we can determine whether he reacts in a physiological or pathological manner to his environment.

On admission, the physical examination of H. B. was negative, except that the pupils were seemingly myotic,—reacting very slowly to light and on accommodation,—while the patellar reflex was very slight. These variations from the average were not greater than are occasionally found in individuals in whom there is no suspicion of organic disease of the central nervous system; and, as frequent subsequent examinations failed to disclose other

[1] See "Paranoia, Systematized Delusions and Mental Degenerations," by J. Siglas. Translated by Dr. Wm. Noyes, Journal of Nervous and Mental Diseases, March, April, May, June, 1888; also "Litigious Insanity," by Dr. Edward B. Lane. Trans. American Medico-Psychological Association, 1902.

neural anomalies of significance, the conclusion seemed justifiable that the apparent myosis and diminished knee-jerk were normal to the individual. It ought to be noted, however, that the opposite condition,—a dilated pupil and exaggerated patellar reflex,—is more common in patients of a neurotic organization, to which class H. B. clearly belonged, and that a spastic myosis is about the most common eye symptom in paresis. The mental examination was continued for from one to two hours at a time on several consecutive days and the first impression was strong that we were talking with a victim of early paresis. Paretic, paranoiac or moral imbecile seemed to be the diagnostic question.

Our patient manifested quite enough egotism for a paranoiac. He was perfectly coherent and had a good command of English, but his memory seemed poor, in that his long story would differ in some essentials from day to day and there would be many inconsistencies in his statements of alleged facts. "Oh, I do not claim to be a lightning calculator," he would say, when his attention was called to the fact that his dates were badly awry. Our case records say: "His story is very disconnected, either because of a disturbed brain or because he is lying so rapidly that he cannot maintain the proper sequence of events. Any discrepancy brought to his notice he immediately cleverly explains, but in a way to vary his original stories considerably. The good nature and optimism which he shows, the ease with which he speaks of large amounts of money and extensive commercial interests, his good-natured egotism, the many discrepancies in his stories, the readiness with which he explains, though not always satisfactorily, together with some suspicious physical signs, the contraction and slight mobility of both pupils and almost absent knee-jerk, give rise to a strong suspicion of paresis."

This picture certainly does not suggest the typical paranoiac, if we use the term in the restricted sense of the "original monomania" of Sohmer and the "primary monomania" of Spitzka and Kellogg. The paranoiac's memory is commonly good, often preternaturally good. Morbid introspection has seemingly fixed every event of his life in self-consciousness and so exaggerated its importance that each personal experience for a series of years is faithfully reproduced as to dates and consistently as to sequence of events.

With the paretic the symptom-complex is strikingly different. Here we have an organic disease of the brain, a chronic meningitis with atrophy and softening of the brain cortex, the product of "civilization and syphilization," as Krafft-Ebing has tersely put it. A good-natured dementia is the leading characteristic. Grandiose accounts of his experiences,—apparent mendacity,—are the rule; petty acts of dishonesty are very common. A jolly good fellow and a spendthrift, a lover of good wine and bad women, in the early stages, frequently a petty criminal later on, almost surely bedridden in four years, with far advanced motor and sensory palsy, he yet insists that he is "all right," that he has lots of money, hosts of friends, many wives, perhaps, and that he never felt better in his life.

The imbecile is of another kind, for his troubles are many and his chief mission seems to be to make trouble for others. That particular type called the moral imbecile,—because the moral sense is most conspicuous by its absence—has been well called "first cousin" to the paranoiac. It is doubtless true that a bad heredity,—an unstable nervous organization, a vice of constitution,—is the soil from which both have grown, yet there are important differences.

H. B.'s previous history, as related by himself, was so long that he was asked to give it to us in writing, hoping to get an abridged edition. The written autobiographic statement was "toned down" a good deal from his verbal statement, and I can only include enough to illustrate the nature of his statement and the writer's coherence of thought.

FEBRUARY 5, 1902.

Gentlemen:

In accordance with my arrangement with you, I herewith send you a brief statement relative to certain transactions and facts related to you verbally. I am the son of M. B. of London, England. My father was owner of the City of London Printing Works at one time, also owner of the Rotary Ticket Printing Machine (patent). My father was a promoter, financier and private banker in London and has successfully floated many large companies. I was informed that my father had purchased and brought out the International Cable Company, the Bermuda Cable Company, the Quartz Hill Gold Mining Company, the Parcherry Mining Company, the Wala Wynad Mining Company, the Santa Rosalia Mining Company, the Washington and Seattle Breweries Company, the Iowa Pacific Railway Company, and many other large companies too numerous to men-

tion here, but which will be more particularly set forth by my relatives and others. . . . I am a lawyer, studied law in London, England. Educated in Dublin and London. Was manager for M. Barn & Co., Law and Mercantile Agency, New York. Was manager and president at one time of the Joseph Jackson Company, incorporated, at 258 Washington St., Boston. Was manager for the Rollins Harlow Company at No. 1 Beacon St., Boston I was in partnership with Frederick H. Jackson, Real Estate Broker, 23 Court St., Boston, Mass. I am informed that the said Frederick H. Jackson is a man of means. I have lived at Wellington, Massachusetts, and kept two horses and carriages and owned my own furniture there. I lived at 321 W. 14th St., New York City, and owned furniture there and valuable paintings. I am perfectly willing to make any statements written or verbally as to the above facts and you have my full permission to write to the parties above referred to. (Signed) H. J. B.

This rather incredible story written with care, without the embarrassing factor of a cross examination, was not made wholly of imaginary "mind stuff," as are the roseate ideas of the general paralytic. He was the son of a once wealthy Hebrew whose name and address he correctly gave. He was petted and very much indulged as a youth, as will be shown by other evidence. The proportion of truth and falsehood in the history, as a whole, could be fairly judged from the mother's letters which have an intrinsic value of their own besides throwing a flood of light on our patient's peculiar mentality. It will be admitted that the absurd statements of the paretic and the persecutory and expansive ideas of the paranoiac are sincere beliefs, due in the one case to active brain disease, and in the other class to a slowly progressive psychical degeneration. On the other hand, many sane people delight in "drawing the long bow" and the talent for weaving a thread of truth into a fabric of falsehood so cleverly that the single thread will first catch the eye of the casual observer is a characteristic accomplishment of the moral imbecile, and the habitual liar whose sanity is not usually questioned. When cornered by a cross examination, H. B. would admit that some of these many companies successfully "floated" by his father were "brought out" on paper only, and I would here observe that nothing more clearly marks the difference between the absurd fancies of the paretic or the systematized delusions of the paranoiac and the "fish stories" of the imbecile, than the efforts of the latter to support each falsehood with two more of a little different shade.

A series of letters, remarkable in some ways, was received

from the patient's mother. These letters, to my mind, are of sufficient psychological interest to be read in full, but in a reasonable time limit I can only quote a paragraph here and there. In her first letter, dated February 7, 1902, she writes: "Your letter to my husband was handed to me. I do not know as I shall give it to him, as he is at present suffering from acute heart trouble. In the meantime I shall be perfectly explicit with you, although I am not filling up your enclosed form. My son is of ill balanced mental and physical organization, neurotic, hysterical, of strong passions, weak moral force (in fact of no moral fibre), no sense of duty, a victim of an exaggerated form of intellect which causes him to see and judge matters as a horse's eye sees an object. He is clever and artful and in him the disease would be moral insanity. A sister and brother of mine are in the asylum. Our unfortunate son was not properly brought up. His father showed him an extravagant example and would not have him taught bodily work. He was indulged in every whim, as his father is a man with no sense of responsibility."

A second letter, dated February 28, 1902, gives added information of interest and further illustrates the mother's mental organization. She writes: "Anything you ask shall be answered per return mail. . . . My son's affliction did not spring *ab initio* from any involvement of neural processes, although through hardship injury may have subsequently occurred. I assure you that instead of his conceptional faculties being impaired, to my mind they are vivified. Strange to relate, more like a fairy tale, every word he related, as you have written, respecting his father and himself, is truth, but in the abstract. In detail, they would admit of a very different construction to what he would have you understand. He has not drawn on his imagination for the events of which he made you acquainted, but he is either mendacious or misled as to results. You asked me at what period I noted a change in my son's demeanor. He was always obstinate and ungovernable and when at a very youthful age he consorted with bad companions of both sexes, then the influence of a mother was quite overthrown. Mind, he seemed to be possessed of great ability and great industry and huge capacity to work in what occupation suited him. Here we get great force misapplied producing *crime*, as it invariably does. His defects are congenital and the law of heredity in

this instance is painfully substantiated. His, notwithstanding his great capacity, is a low order of human intellectuality. Two senses fail him to balance the overwhelming forces of his nature. *Sense of danger of the law of results and sense of responsibility.* Admitting this, then where can the poor human vessel look for ballast? In palliation and extreme grief for the strait he has fallen in and the awful result of the 'sins of the fathers,' etc., in pity bear in mind that he had an over-indulgent father, who had not the capacity to control him or to show him a good example to restrain him. I was adjudged a hard mother because I wanted to spare the child and not the rod. As this tended to break the peace and disturb the rest of the household I was bade to desist. Hence the result. . . . They tell me that I am a pessimist, that unhappy being whom everybody wants to avoid for the sake of the bliss of living temporarily in a fool's paradise. It may be true. Can I help it? . . . Have I not been deprived of the power to enjoy? I am resigned, for an Almighty Power animates me to throw myself into the breach and save those that would otherwise be lost. I have brought up ten children credibly (except the eldest). All my daughters are exceptional honors to me. My husband's businesses have failed and we are in financial straits. I am at the helm and dare say will steer through all right with Divine help."

On March 28, the mother writes again: "As you see him now so he has always been, hounding me for money to gratify his whims to squander it. On the other hand, from his writings, etc., he is not a fit subject to be in a lunatic asylum. In justice I must say this, because people are at large who are not so logical or constructive as he is. If people with an *idée fixe* were always confined, the world would be one huge lunatic asylum. Although he is morally obtuse, and although *in* the world not *of* the world, merciless, ungrateful, unnatural and unprincipled, without the slightest force of natural instinct, morally a monster, still he is not what many thousands would pronounce insane. We know he is the victim of an obscure brain disease. Many times puzzled, I often think it is malformation of the brain, with strong passions, and no moral force to govern all. . . . He was always litigious. Of course there is policy and method in this, for when a swindler is discovered he relies on his clever lawyer to find a technical point

for him to elude conviction and consequences. This is all to me something too shocking from my point of view and really my brain seems to be giving away. I feel as if I had been smelling nitrate of amyl. I am an old woman and shall be getting brain lesion with it all, no doubt paralysis. My nerves and muscles all ache. I am fast losing power of volition. What shall I do?"

While physicians may smile at the mother's analysis of the case, I think members of this society will agree that her observations were keenly intelligent and her deductions not very wide of the mark. If one wished to write a thesis on the relationship between genius and insanity to prove Dryden's oft quoted clause, "Great wits are sure to madness near allied," the forceful and pathetic letters of this highly gifted Jewish mother would serve as strongly presumptive evidence. The mother writes again on April 10: "It is not that Harry is so litigiously mad. He is revengeful, it is true, and craving for notoriety. What he really is, is a blackmailer, a past master in the act of insinuation and blackmailing. When he goes to New York he will commence the blackmailing profession over again and get himself run in again. No, Mr. Drew, I will not undertake the onus of accepting the charge of him. . . . It would be better for me to go to the devil than to bring the devil upon me. . . . I wonder if you read his one part deceitful letter, the other part as impudent, arrogant and overbearing as ever."

Apparently the mother's high tension organization had been taxed almost too far by the son's importuning letters covertly threatening, if she did not send more money, to publish his grievances in the newspapers, using his own notorious record as a weapon to extort money from his family through their natural fear of a sensational publicity which would certainly injure the other children and further stain a respected family name.

Twelve days later she writes again, and now, the mother instinct being again dominant, she argues in defence of her son that he may not be returned to prison, even though she cannot have him sent to an asylum in England, as had been proposed. Both explanation and argument are so creditable to the mother's heart and intelligence, and so interesting from a psychological view point, that I venture to quote further from a letter dated April 22: "While I thank you for your goodness in endeavoring to

get Harry sent here to an asylum in London," she writes, " I dare not bid him to be brought over. Did I do so it would at once break upon the home and scatter its inmates. My daughters are working might and main at honorable professions to help me to get a living for themselves. One of them is engaged to an influential man here. My husband has only to receive a shock,—he will die. Therefore they object to my bringing over sure trouble. They affirm that Harry would not rest in an asylum here, but would insist and succeed in making himself heard to air his grievances. The newspapers would ring with the unusual news, the members of my immediate family, where they were previously respected, would be surely shunned, no doubt their professional engagements canceled, my husband killed, myself paralyzed by despair, and all for what? To save the feelings of one unworthy refractory being. How can I contemplate the commission of such a crime? I dare not do it. Mr. Drew, that man cannot be sane. Nor is he. It may be true that were he to speak in his lucid, able and logical way to even experts in insanity they would not detect the flaw but declare that only a man in possession of his senses could so ably argue and manipulate documents and seize on technical points of law to get in the thin edge of the wedge to let himself out. They might not agree that his is an obscure form of dementia, a case, to dive deeply, clearly and concisely into it, where the objective mind is more or less clouded or impeded whilst the subjective mind is permitted a force and liberty which could not belong to it were he in a condition of complete sanity. With his avowed declaration to fight *law* and *power,* to go to New York to blackmail Chapin, *regardless* of consequences, even any medical man decided to maintain that he is as lucid and logical and clever as any other man on the same plane, if not even more so, must at the same time admit that there is a departure from the normal balance and recognize his mental irresponsibility. The man has gone mad. His subjective mind is sole and supreme in the display of its functions. Thus—what is it we perceive? Why, a recollection of one rooted experience, one grim resolve, the *idée fixe* of the monomaniac, which will possess and play the tyrant with his mind, cause him to pursue his suicidal purpose to the bitter end, always regardless of consequences, as unreasonably and fatally as the moth flies in the flame."

The contents of these letters are my best excuse for the lengthy quotation. The son's style of writing is strikingly like his mother's. The following to his wife was written on one of those days when he was apparently on good terms with her, but he was a master of blandishments to obtain money or gain a fancied vantage.

March 12, 1902.

My own darling wife:

Just a few lines to tell you I received a letter from my mother on Monday and it was enclosed to Dr. Drew. I understand my mother's meaning and everything is satisfactory. When you come to see me on the 26th of March I will give you a draft for some money and I will explain things fully to you what to do. I shall draw on a certain person at sight and the money *is to be paid to you* and *you alone,* as you are my wife and I cannot trust strangers. I shall not call upon you to pay any one else but Mr. Fishacher, yourself and myself. I have received some very satisfactory letters from New York and Boston. I cannot write it. Everything *is going well.* Cheer up, little wifie. God is helping us. I have had some blank drafts sent to me and I shall have a letter all ready for you and draft on the 26th of March *positively.* Write to me at once and tell me all the news. Write to my mother again and tell her not to write to Dr. Drew again. Be careful what you and Mr. Fishacher say to Dr. Drew. I shall explain all when I see you. I have some startling and good news for you. I drempt last night of you all and of Queenie playing the piano and Clarence. All you, my three darlings, were kissing me and loving me. I am lonely at times without you, dear. You said that our children would write to me last Sunday. I have not received any letter yet. Write often to me and cheerful letters as it is good for me. I am taking fresh air as you wished. Your poor husband is battling against breakers but he will come into Port and although tossed and wounded on the ocean of life sees the Lighthouse not far off. Our ship will soon sight land, my wife. Cheer up. God bless and guard you all.

Your loving husband,

(Signed) H. J. B.

P. S.—Good night, love, and kisses - - - - - -. Be sure and be here on the 26th of March. Write me you are coming, Love. - - - - - - - - - -

The following to his mother was dated March 28, 1902:

March 28, 1902.

My own beloved mother:—

Your letter duly received enclosed again. I again ask you to write to me direct to Mr. H. J. B., State Farm, Bridgewater. It is not necessary to put word Asylum. Now let us come to some common sense talk. I agree with you it is bad to be robbed by lawyers. Recapitulation, recrimi-

nation & transportation seems to be your peculiar mania & writing chronic lovely incriminating epistles about *insanity* in my family and such untruths & foolishness, & giving Dr. Drew & the State Board of Lunacy the power to hold me *indefinitely*. You are trying to bury me alive & turning the key on me with double padlocks. Clever muddlers. I had arranged every thing well. Mr. Fishacher and my wife came on the 26th of March & in the presence of Dr. Drew my lawful wife said the words, "Dr. Drew, I have come for my lawful husband, his sentence has expired, and I, his lawful wife, am here to take him into my custody." Dr. Drew replied, "I regret, but I must have an order from the State Board of Insanity." Now the State Board of Insanity is a tool of Dr. Drew's and it is simply a see saw formula game, exposed in the newspapers before, robbing men and burying me alive in a living tomb. However, Mr. Fishacher has arranged with the Commissioners to give me a ticket of leave and the State Board of Insanity must let me go if my wife, who is my lawful *legal* guardian, takes me in her custody. Everything is arranged and poor Gertrude is on deck ready. We shall not make a demand until the $250.00 is here. Send the draft to Gertrude at once. Do not muddle matters for God's sake. I shall follow out your ideas in many ways. Remit to my wife, Gertrude B., at once. I am satisfied she is faithful and loving. She lay in my arms all day and is pining to get me back with her and the children. Do not delay a moment. Leave all to me. Mr. Fishacher and Gertrude & myself will arrange everything. On receipt of the money, wifie will get me a suit of clothes, some shirts & pay my fare and I shall come out all right. The reason why I want the money at once in my wife's hands is because when she sees the Board of Insanity with Mr. Fishacher she will state that she has money to take care of me and that I will not be a charge on the state & Mr. Fishacher has got the ticket of leave ready from the Commissioners & Police. Remit to Gertrude at once. Do not muddle me into my coffin, as that will be the next step, my coffin. You have all muddled me into a madhouse & the next will be death. Stop muddling. You have a dangerous pen writing about insanity. The insanity dodge adopted by lawyers is good in some cases, but not in all. Use tact and judgment. Stop this ghastly work of thwarting my able efforts for release. Remit & you will soon get a cable from me when I am *free*. Gertrude must have money in hand to state she is able to take *me*. Do you understand or are you obtuse. When you wanted money I sent it to you. I need help now from a lunatic asylum. I know, my own beloved mother, you mean well but it is a case of life & liberty & I must speak plain. Be sure and remit at once. Instead of your coming over, my wife will do your work for you and take me. Send her the money. I can be out now in an hour but she must have cash to say I am all ready & not a burden to the state, etc. I am now at your pleasure, waiting in the madhouse for funds for help from you. Cable, remitting. May God bless you & keep you & *guide* you. I shall find lots of work to do & will be a good faithful father to my

darling children & a good husband & a faithful son to father & you. I long to kiss you both again. Good bye.

Your loving son,

(Signed) H. J. B.

P. S.—Remit cable. Remit to Gertrude, she is faithful & true. I have found it out. Remit. Waiting. Help. I shriek a sane man from the mad-house. Help.! Help.! Help.! Remit, cable.!

Another letter to his wife, throwing light on his mental content in an angry mood, was as follows:

APRIL 10, 1902.

Written under duress.

ORDERS.

To Mrs. G. B.

(1) You are hereby ordered and directed by me, your lawful husband, to bring any money you may receive from Mrs. C. B., my mother, or from any party or parties on my behalf, to the above Lunatic Asylum and to deliver the same intact to me and you are forbidden to remit by mail any money to me or deliver any money to Dr. Drew or any person or persons on my behalf. You are ordered to come yourself or send Queenie G. B., my daughter, with the said money, but not to entrust the same to any other person or persons.

(2) You are ordered to cause Queenie G. B. & Clarence W. B., my children to write to me forthwith and to have the letters here by Saturday, April 12th, 1902.

(3) You are ordered to go to the State Board of Lunacy at once and make a legal demand for the father of your children's immediate release from unlawful custody.

(Signed) H. J. B.

A copy of the above orders has been sent to *my* Attorney, Mr. Fishacher, Esq.

The foolish arrogance of these "Orders" might come from a paranoiac or an imbecile, but the paranoiac, if educated, has commonly more sense than to write such stuff to any one. The following explains itself:

FEBRUARY 21, 1902.

To the Manager of the Pinkerton Detective Agency, Boston, Mass.

Dear Sir:—

Upon receipt of this letter please send a reliable trustworthy and shrewd detective to see me. Tell him to bring blank drafts with him. I was robbed and unjustly sentenced to Massachusetts State Prison and two months before the expiration of the sentence two unscrupulous quack

doctors have conspired with Officials and railroaded me to a Lunatic Asylum. I complain of foul play. I will not write full particulars but will explain fully to your man. I have received a cable message from my mother in London, England, and my friends will be here soon. I am engaging reputable lawyers to reopen my case and to get an investigation. I am a Lawyer. I have startling disclosures to make to your agent. Please make your charges as reasonable as possible and remember I am an attorney and may need you for many cases. Send your man at once and on his arrival I will pay him a good substantial fee. I have powerful friends. I have been sent to an Asylum to save the Officials from exposure, etc. Foul play. I cannot send any remittance by mail, as I do not want anyone to know who I am drawing on or where my money is, owing to the trustee and attachment law being so strict in this state, and as I have legal trouble with my wife and she is looking to get alimony and to attach my properties &c. and the Officials here would know where cash is if I drew through them as I should have to get a draft from them. Send good man with blank drafts. I will pay his fare and all expenses. Send as quickly as possible and be sure he sees me and tell him to be silent as to who he is. I need your man to hunt up evidence, &c. &c.

Yours respectfully,

(Signed) H. J. B.

This letter to the Pinkerton Agency, it may be noted, mentioning "legal trouble with my wife," antedated his loving letter of March 12 by eighteen days. He would not forget to write "bring blank drafts" in each of his many letters to lawyers, evidently thinking this suggestion best calculated to bring the lawyers quickly, and usually in his letters to the latter he promised payment in full for past services. When one lawyer came to see him, our patient did use the blank drafts brought as suggested, but he drew on another lawyer with whom he had formerly been on a friendly footing. This lawyer friend had also been promised full payment if he would come from New York City to co-operate with Massachusetts attorneys to obtain our patient's release. He had also been admonished to bring blank drafts. This New York attorney was evidently slightly indignant to find that our patient had drawn on him in favor of another lawyer. Some letters were exchanged, which we did not see, the Boston lawyer, who was paid by drafts on the New York attorney, acting as mail carrier for our patient. The following letter was written to the New York attorney, in reply, evidently, to a sharp letter from the latter, who had discovered that his some time friend, our patient, was not acting on the square with him. It reads as follows:

My dear Dave. MARCH 27, 1902.

Your letter duly received and contents noted. Your extraordinary epistle and the peculiar tone & indignant manner in which you write can only be appreciated by me, as I understand you are a fighter and an indomitable *toner,* and I verily believe you would rather fight than eat and your bickering & quarreling with me, your own client, is proof to me that you are a very accentric Attorney in some small matters, but with all thy faults I love thee still, & look upon you as the faithful *wondertoar* young & reliable checkmating General that you always were. I mean you try to checkmate my enemies in a legal manner. Let me explain a little. The fact that you say you never received a pawn ticket for ring & stud shows me that you have never received it but I solemnly declare that I delivered it to the prison officials with a letter addressed to you, stating that the ring & stud had nearly run out & sent it to you as a present if it would be any use to you. This is over four years ago & if you have never received it then it only adds one more crime and felony to the list to be laid at the door of the unscrupulous Prison Officials & I am not to *blame* and you are not to be blamed. - - - - - - - - - - Now let it be distinctly understood that I am not blaming you in any matters but merely wrote you the other day because Mr. Bruorton stated that he had received a letter from you and that you asked him to go to the Asylum and find out if I had the money to send to you for expenses and that you would find the affidavit drawn & sworn to if he desired you to do so & Mr. Bruorton stated that he wrote you that you could use your own judgment as to the affidavit, but would not be responsible for the costs or for your expenses or for any work you might do for me. Upon receiving such a letter I immediately wrote to you to look at the past &c., &c., as I think, in view of the past & taking into consideration that I owe you nothing & that I have always faithfully and honestly remunerated you for your services that, to an old client, you need not be so particular and parsimoniously inclined. Mr. Fishacher, a Boston lawyer, was here yesterday with my wife and she stayed all day and dined with me. I delivered a letter to my wife to have registered a draft &c. to you. Please come on and see this man at once on receipt of the cash from his brother. His brother's address is on the draft. In the meantime if any money arrives, I shall telegraph you $50.00, as I did before through the Western Union Co., as I have received a heartrending letter on Monday from my father & mother who state they are sending me *money.* Have faith in me, my brave Legal Warrior, & be patient & do not write recriminating epistles acting as an injured man when you are not. You have a peculiar faculty for quarreling. Do you remember the order of Judge Newberger dangling in mid air and Judge Pratt's stays (while you wait) and the conflicting orders and toners moving mysteriously as if by peculiar power & Severance supplementary orders and Referee Hess and Referee Stein being removed. DANGEROUS on the other *side.* Look out I am coming soon. I rest my case.

Your affectionate friend and client.

P. S.—Write soon. (Signed) H. J. B.

It would seem, on the face of it, that a lawyer who would try to work the confidence game on his professional brothers must be very insane, and it ought to be known in this connection that most of the attorneys to whom our patient wrote either did not notice his letter or else inquired by telephone or letter of the Asylum officials concerning patient. Attorney Fishacher knew the man and was trying to serve him, for his family's sake, in an ethical and honorable way. Another Boston attorney seemed to swallow his bait whole, made frequent trips to the State Farm, came and went, as a rule, without consulting with the Asylum officials, until he obtained our patient's draft for $1800.00, $300.00 for himself and $1500.00 for a New York law firm as whose agent he professed to be acting. After this his visits were infrequent and we do not know how much he realized on those drafts, nor how he felt when he heard from that New York attorney on whom the drafts were drawn, and whom our patient addressed as " My dear Dave." The conclusion was forced upon us that our patient was not a true paranoiac or monomaniac. I will admit that, using the term paranoia in its broadest generic sense, it may include most kinds of mental deficiency outside of dementia paralytica and the acute psychoses embraced by the manic-depressive syndrome. I do not forget that Dercum[2] and others would include under paranoia all cases of chronic systematized delusional insanity, and that Ziehen writes of stuporous and incoherent paranoia. Dercum's classification seems eminently rational and helpful, if we agree as to the limitation of terms, but we hold that our patient's so-called delusions had not the true ring, and to speak of " stuporous " and " incoherent " paranoia is, seemingly, to confuse paranoia with dementia præcox, or a toxic insanity with confusion, else to give a class name to a late and unusual phase of a disease. We believe the phrase, " constitutional inferiority with moral perversion," best covers our patient's abnormality. The direct question, " Was he insane? " cannot be properly answered by yes or no. There is no doubt but that he knew the difference between right and wrong in the abstract, and with reference to every dishonest act he did, but this could be said of almost all paranoiacs. The weight of evidence seems to show that he could

[2] Journal of Mental and Nervous Diseases, September, 1901.

act or refrain from acting according to his judgment of which was the best policy. The fatuous folly of his judgments is beyond dispute. Insincerity, duplicity and mendacity were characteristics evidenced at every interview and in every letter he wrote. To the Governor and the British Consul his letters were couched in technical and well turned phrases. To the lawyers he emphasized the fact of his being a Jew and of having powerful friends and rich relatives. To these he wrote of being robbed and illegally sent to an insane asylum, but to his mother he wrote very little about being robbed. To her he cried " I am hungry and abused. Help! Help! I shriek a sane man from a mad-house! " We know this was not delirium. It was carefully penned,—all the exclamation points were in place. The motive was, manifestly, to touch the maternal heart, to work on the mother's nerves that she could send more " geld " quickly. There was just a little basis for his claims of being robbed, but not by any prison official. A certain New York lawyer had obtained money from the patient's mother, more than one hundred pounds, ostensibly for her son's defence. There was an effort to get more money, as the attorney's letters, which the mother forwarded, plainly evidenced. Our patient knew this, but it suited his purpose to make charges against state officials, because these charges received attention. To charge lawyers with getting money from his mother by misrepresenting the case, would avail him nothing. People would laugh at that. May I requote a few significant lines to " dear Dave," who resented being drawn on in favor of the Boston lawyer. " I verily believe," H. B. writes, " you would rather fight than eat & your bickering with me, your own client, is proof to me that you are a very eccentric attorney in some small matters, but with all thy faults I love thee still." This illustrates our patient's cleverness at cajolery, and counts against paranoia, because a sense of humor is almost as rare to the paranoiac as a sense of honor to the moral imbecile. It is difficult to conceive of a true paranoiac " jollying " another about anything.

The more we studied our patient, the more it seemed to be true that his mistrust of every one,—his " delusions " if you please,—were not the delusions of pyschopathology but rather false beliefs based on his peculiar experiences and his own self-consciousness. He had little knowledge of the reactions of an

honest man's mind. The altruistic sense and the sense of honor were rudimentary, but these are the products of man's higher evolution and are probably rudimentary in most habitual and instinctive criminals. The primordial instincts of self-preservation and love of offspring seemed fairly developed, albeit his faulty judgments and ignorance of men and institutions as they are made his foolish pertinacity in wrong-doing seem suicidal. Read "Hard Cash," by Chas. Reade, he would urge, in defence of his ideas. "Hard Cash" had apparently shaped our patient's beliefs and destiny in more than one sense.

After we had studied H. B. for more than three months, I advised that he be returned to the prison from which he was committed. He was accordingly returned to prison, as per Section 101, Chapter 225, of the Revised Laws of Massachusetts, and held for about seven months, during which time there was no essential change in his condition. Early in December his mother came from England and was allowed to take her son, as his minimum sentence had expired. We were informed by the prison physician that his mother insisted, to H. B., that she had all the legal knowledge necessary to free him and would not allow him to talk. "She was the only mortal who was able to control him in this respect," the physician added. It was understood that his mother proposed taking our hero to South Africa. The inference was not that the talented mother bore malice against that unhappy land, but rather that no indictment stood against our sometime patient there.

"If this man should kill someone would you hold him responsible?" was pertinently asked by a physician whom I advised of my purpose to recommend the return of H. B. to prison. Being persuaded that there was no pathological condition present that would compel him to commit a homicide, I would hold him responsible as I would hold another weak-minded brother responsible. Not absolutely responsible, in a moral sense, nor entirely irresponsible, as the man suffering from brain disease, but sufficiently responsible to be kept in prison. Too much responsible to be allowed to prey on the truly insane. (H. B. was constantly working his "confidence games" on other patients. Stationery, fruit, and delicacies he wheedled others out of daily, paying generally in fair promises.) It is true that graded responsibility is debatable in

medicine and not recognized by the courts, but we are not entirely responsible for the defects of the courts. The natural instinct of civilized man is to save life,—his own life first, then others. This order may even be reversed, in the highest types, in favor of the nearest and dearest by ties of blood. So it is that the instinct to kill is against nature and raises the presumption of psychopathology. The acquisitive instinct, on the other hand, is according to nature and an exaggeration of this instinct is presumptive evidence only of moral deficiency, even to the making of a "common and notorious thief." We do not know that H. B. had any impulse to kill or do bodily injury to anyone. His mother's testimony was to the contrary. Moral imbeciles are often exceedingly cruel and enjoy beating, cutting and killing for its own sake or for the sexual frenzy excited most intensely,—so to speak,—by the taste or smell of blood. Dr. Howard's heroine (?) Mizpra,[3] was considered entirely irresponsible. Possibly so, from a purely academic viewpoint. It is doubtful if the courts would support the contention, and practical psychiatry might classify the case as not amendable by hospital methods. It was very clever of the author, therefore, to have Mizpra hurled into a yawning abyss before she reached an insane asylum.

To glance again at the mother's analysis of H. B.'s case, is it not reasonable to assume that the man's objective mind was in abeyance, that the subjective mind held supreme sway, forcing the individual on in his irrational course "as fatally as the moth flies in the flame?" Was his oft declared purpose to fight his enemies in the courts,—"to pursue them with the dog whip of the law and hang them on the gibbet of public opinion" a manifestation of the "one grim resolve, the *idée fixe* of the monomaniac?" Concerning the subjective mind hypothesis, a review of much of Thomas Hudson's[4] dual mind thesis would be needed for an adequate consideration of this phase of the subject. I will only observe that if the "subjective mind" is identical with an immortal soul, as Hudson holds, and is responsible for the atrocious acts of some persons on this planet, then the ancient theological conception of an unquenchable lake of burning brimstone

[3] The Perverts, by Wm. L. Howard.

[4] The Law of Psychic Phenomena, by Thomas J. Hudson.

seems to have a justification. As to our patient's passion, for litigation being the fixed idea of a monomaniac, much more might be said than time will allow. A love of litigation may be but a manifestation of a quarrelsome disposition which, unfortunately, is not limited to the insane. So far as it is an evidence of insanity, it points to imbecility quite as much as to paranoia. The same may be said of inordinate vanity and love of notoriety which is nourished by the publicity of the courts. H. B. might have engaged and discharged a dozen lawyers while in prison for the mere love and excitement of it. It is probably that he did not intend to pay any of them, except by promises and bogus drafts. Experience had taught him that letters to lawyers and State officials were forwarded. He was a forceful writer and a ready talker and loved to do both. Most of all he longed to get into court to show off his talents before newspaper reporters and a crowd. When pressed on cross examination, concerning the false charges he was reiterating against the prison officials, he once declared doggedly, " A man has a right to defend himself." This was the key to many of his " delusions,"—created for a purpose. He believed all men to be dishonest tricksters,—this was a natural subjective reflection,—and he was doubtless disgusted to find so many lawyers who did not think as he did. Life in " Cherry Hill " was rather monotonous, why shouldn't he keep on engaging attorneys till he found one of his own kind, who would help him into court or get his letters published in the sensational newspapers? He was stubborn and opinionated,—like many weak-minded men,—and some others; his little knowledge of the law puffed him up and made him intolerant of advice, but he was not dominated by " one grim purpose; " on the contrary he was vacillating as most men, at least. To-day he knew his wife was true and loyal, tomorrow he was sure she could not be trusted in anything; this week he was willing to follow the advice of his mother and attorney Fishacher, the next week his only reliable legal advisor was the Boston attorney who was paid by drafts on the lawyer in New York. It does not seem wise to generalize broadly about these mentally inferior and morally perverted ones. Each case must be decided on its own merits. One might strongly defend the postulate that all instinctive and incorrigible criminals are insane, but practical considerations would forbid that

these mingle with those suffering from brain disease. Indeed, the doctrine of limited responsibility is not incompatible with the administration of a modern humanely managed prison, and it ought to be known that the isolation building for incorrigibles called "Cherry Hill," at Charlestown, has very large rooms, larger than any single rooms I know of in an insane hospital, well ventilated and well lighted through the roof, with good plumbing, running water and a good bed in each room. Humanity has a right to expect similar provisions in each penal institution, for the line of demarkation between sanity and insanity is particularly shadowy in the criminal classes, and it is better that asylum features be introduced into prisons than that the hospital and asylum standards should be lowered by prison necessities.

I would express the opinion, in closing, that the committing physicians were fully justified in giving H. B. the benefit of every doubt. They were supported, too, in the diagnosis of "litigious paranoia" by the teachings of some of the best authorities. My specified and implied reasons against the diagnosis run through this essay. I wish only to repeat that while paranoia and constitutional inferiority with moral perversion (moral imbecility) have much in common, may indeed by well called "first cousins," they ought to be separated clinically as readily as hereditary ataxia and locomotor ataxia are recognized as distinct disease forms. The parallel would not hold as to anatomical pathology, because these spinal degenerations have a clearly defined and well-known pathology, but that paranoia is a veritable psychical disease-entity is evidenced by its distinct and fairly uniform stadia. It is essentially progressive, even though chronic, characterized by systematized delusions and hallucinations, especially of hearing, in the majority of cases. The systematic evolution, the distinct period of introspection and depression, followed by a period of delusional exegesis of his own morbid subjectivity, and the hallucinations which may begin to spur him on, followed again by an expansive and ambitious period, with its change of personality,—these are the landmarks by which we may know the true paranoiac. The moral imbecile is not so. He has false beliefs enough, certainly, but they change "with every wind of doctrine," like ordinary weak mortals. He has no period of depressive introspection. What little sense and judgment he has, he keeps to the end of

the chapter, barring the ordinary wear which all flesh is heir to. It is true that the moral imbecile may be far below the intellectual plane of the paranoiac—the differential diagnosis does not rest on the degree of mental defect. That the imbecile may be so defective as to be wholly irresponsible goes without saying. But call his condition "moral insanity," "the insanity of degenerates" or "phrenasthenia," if you please, yet, in the absence of substantial evidence of brain disease, the natural criminal product of a neurotic organization and a wrong education hardly fits the legal conception of insanity. When the impulse of the perverted imbecile is towards rape and murder, as it so often is, the common conception of insanity should be strenuously opposed before opening the asylum doors to let the perverted one free, but when inordinate vanity and infantile judgment leads the individual habitually to seek gratification in the publicity of the courts, the hospital physician's duty to society may not necessitate a rigid adherance to medical[5] as against legal conceptions of responsibility. As the morbid man, bent on suicide by hanging, hardly ever shoots himself, so the imbecile seeking gratification through the courts rarely gratifies his ureasoning anger by personal violence to the object of his spite. Therefore it is that the weak-minded litigationist may be less harmful at large than among the insane, than among other insane, if you choose to have it so expressed.

NOTE.—Several weeks after this paper was written, and less than five months after our hero was released on parole from the State Prison, letters began to come from J. B., from New York City—registered letters, some of them—notifying the asylum officials that legal proceedings were about to be begun for the release of several inmates of the Massachusetts Asylum for Insane Criminals. Letters came from other "counsellors at law" in the same tone and spirit, giving notice of legal proceedings about to begin, "having been retained by Mr. J. H. B., etc." From these letters and other sources we learned that J. B. was "highly prosperous," "working day and night," "employing several lawyers," "supported by the most prominent people in our city (New York) who speak in the highest terms of his (J. B.'s) indomitable business acumen, responsibility and strict honesty." One of these assistant counsellors at law, J. B.'s "brave legal warrior," "Dear Dave," subsequently came on from New

[5] Peterson's brief and inflexible definition of insanity—as good as any—reads, "Insanity is a manifestation in language or conduct of disease or defect of the brain."

York City demanding to see certain patients, in the name of J. B. and the Constitution of the United States. "Dear Dave" brought two baskets of fruit to asylum patients who had been wheedled out of their small supplies of delicacies for the benefit and promised favor of J. B., then "under duress," when he should have his "enemies" (?) figuratively on their knees before him. I wish to record this because it seems, on its face, an honest act and a promise kept. So far as it goes, it indicates sincerity, and any evidence of sincerity, considered in relation to J. B.'s history, would strengthen the hypothesis of insanity. It seems that J. B. did not go to South Africa. His mother, we understand, settled the indictments against him in New York, and there is evidence that his "indomitable business acumen" has been exercised on as high a plane as the promoting" and "floating" of the "Iowa Pacific Railroad" and the "International Cable Co."

THE TREATMENT OF THE MORPHINE HABIT BY HYOSCINE.[1]

By J. M. BUCHANAN, M. D.,

Superintendent of the East Mississippi Insane Hospital.

In complying with a promise made several months ago to write a paper on the treatment of the morphine habit by hyoscine, I am cognizant of the fact that this subject has already been discussed in other societies, as well as through the medical press. However, as members of this Association have much to do with narcotic and alcoholic habitués, it cannot be out of place to discuss the treatment of such patients, and in view of the widely different opinions maintained as to the new method of dealing with this class, it is opportune that this subject be discussed at this time.

It is not the purpose of this paper to deal with the causes that lead to the habitual use of morphine; but incidentally I will say the fault is too often with the physician, who first gives the drug for relief of pain without instructions to the patient as to its danger; on the other hand, many use it wilfully for its intoxicating effect, and the strenuous lives of many require such a stimulant to keep them going. In the end, the drug becomes the victor, and the victim appeals to us for relief.

Until quite recently, but little progress had been made in the treatment of this habit, the profession being content to follow the old methods, which have been described as the sudden withdrawal, the quick reduction, and the tapering-off. The first two cause untold suffering, and are even dangerous to the life of the patient, while the last only prolongs the agony. Any one who has had occasion to use the sudden withdrawal or rapid reduction methods, or who has ever read the chapter on opium and morphine in Pepper's System of Medicine, in which this treatment, with its attending horrors, is so graphically described, must surely feel the need of a more humane and rational method of dealing with this class of unfortunates.

[1] Read before the 59th Annual Meeting of the American Medico-Psychological Association, at Washington, D. C., May 12-15, 1903.

I have never used either of these methods, but prior to 1900 treated all by gradually reducing the dose until the amount was small enough to be left off without producing shock. This treatment requires from two to four weeks, according to the condition of the patient, and entails no end of trouble to the attending physician and nurses, by the impatience, fretting and and constant begging for a little morphine on the part of the patient, and when the last dose is given there is always a struggle, with more or less suffering and nervousness. In 1900 a friend who had a sanitarium for the treatment of morphinism gave me his treatment, which is hyoscine hydrobromate, and after a trial we were well pleased, because the patient suffered less, and we were able to accomplish in three days, with but little trouble to us, what it had taken us three weeks to do by gradually reducing the dose.

The particular agency used in this treatment is the hydrobromate of hyoscine, a product of hyoscyamus niger. The dose is 1-200 to 1-50 of a grain hypodermically. It is a mydriatic, a hypnotic, and a cardiac, respiratory and spinal depressant. In large doses, the effects are the same as those of hyoscyamine and atropine, such as variable pulse, flushed face, shallow respiration, dilated pupils, dryness of the throat and mouth, nausea and vomiting, tremor, unsteadiness, muscular weakness, delirium and stupor. The treatment of hyoscine poisoning should be the same as for atropine, viz: stimulants and coffee.

This we learn from the text-books; let me add that the best antidote for hyoscine is morphine and strychnine. If one remembers this, there is no need of ever feeling the least bit uneasy while treating a morphine habitué with hyoscine.

In the administration of hyoscine for morphinism, the dose should be very small to begin with, say 1-200 gradually increased to 1-100 of a grain to guard against idiosyncrasies. The first effect is to cause the patient to sleep, but after several doses have been given, he becomes wakeful and restless, and in about twelve hours delirium will set in.

As a rule the delirium is mild, and the patient will talk rationally, but sees bugs and picks at the bed-clothing, sometimes strips himself to find the bug, or something he has lost. I have noted the same things following the continued use

of bromidia; the mind clears up in a few hours after the drug is discontinued, and we have never seen any bad after-effects from its use.

The following outline of treatment is usually observed, but sometimes the condition of the patient necessitates some changes.

If possible, keep the patient under observation for a few days, and the amount of morphine and cocaine is reduced to what will keep him comfortable, but this is not necessary. The night before beginning the hyoscine, 6 to 8 grains calomel, in combination with some vegetable cathartic, are given, and this is followed by a saline the next morning. The usual morning dose is given, but when the patient calls for morphine in the afternoon, hyoscine is given instead, and from this time for 36 to 40 hours, the patient must be kept under the influence of the drug, 1-200 to 1-100 gr. being given every two or three hours, according to the condition of the patient, but never enough to completely stupefy him. The patient should be kept in bed, and a nurse is kept with him day and night, because all will not stay in bed, and some undress or otherwise expose themselves.

If the patient is robust and bears treatment well, nothing but hyoscine is given, but if he be weak, or if the pulse becomes weak or irregular, strychnine is given, and if necessary a little morphine or codeine is added. The pulse is usually lowered in rate, and as a rule is of good volume, but occasionally it intermits, or becomes weak; in such cases strychnine is indicated, and the heart's action will become normal after the hyoscine is omitted. I have never seen any trouble in respiration, even in asthmatics. Rarely has there been diarrhœa, nausea or vomiting, but occasionally there has been some trouble, when the condition of the patient was not good before beginning the treatment. There is not much appetite, but water and milk will be taken if given by the nurse at regular intervals, and this is important, otherwise the patient will come out of the treatment feeling prostrated. Some become nervous and do not sleep at all during the first nights, and may require a hypnotic for a while; for this purpose trional or chloral hydrate with bromide of soda may be used with good results. If the patient is put on large doses of bromide of soda for a few days preceding the

treatment, or if hyoscine is given in larger doses at shorter intervals, there will be less nervousness, and he will pass the time in a deep sleep. I have had but one patient to sleep through the treatment, and in this instance the hyoscine was given while he was under the influence of a large dose of morphine.

The after-treatment is the same as in other methods, tonics, nourishing diet and rest. Hyoscine will no more cure the morphine habit than will the gradual withdrawal method, or any other treatment now in vogue. No claim is made for it as a specific for morphinism, but it has been well said that "in hyoscine hydrobromide in morphine habituation, we possess a safe, certain and painless method of treatment," and this remedy merits the attention of those interested in this work. It has been said to be inhuman and dangerous, because it produces delirium and sometimes the administration of stimulants, such as strychnine, atropine and nitroglycerine are indicated. I have seen the necessity for stimulation while treating patients by gradually reducing the dose, and have noted maniacal manifestations with hallucinations of sight and hearing.

Now, just how hyoscine acts and takes the place of morphine I am unable to say, but I know it does, and that too without pain or shock. It does not matter how much morphine alone, or in combination with cocaine, the patient has been accustomed to take, he does not miss the morphine after he is once thoroughly under the influence of hyoscine, nor does he crave it after the treatment is finished. I have heard them say they felt the need of a stimulant on account of weakness, but could not say they wanted morphine.

It has this effect without any mental therapeutics—contrary to what has been claimed—for in one instance (Case 2 reported) it was given very much against the man's will. He declared it was a fake and did not want it. He had been treated before by gradually withdrawing the drug, and also had been to a Keeley institute, but did not want any more experiments. He fought against the hyoscine during treatment, declaring that he was badly drugged and often called for morphine during the first day, but his physical condition did not show evidence of shock or pain, and a small dose of morphine, or a hypodermic

of hyoscine satisfied him. The result of treatment was entirely satisfactory to us, and the patient was delighted.

In this matter I think the evidence of the patients themselves is worth something. They do not entirely lose consciousness, for they keep up with time, and can talk rationally about their condition. I have seen them laugh at their delusions, telling me just how they felt, and if they suffered we would know it, for we all know that a morphine fiend will not bear pain without letting us know it. They do complain of feeling weak, sometimes the dryness of the mouth and throat is annoying, and a few complain of backache and other minor troubles, but none of the pains incident to the sudden withdrawal of morphine. I have treated some who have run the whole gamut of so-called cures; a few had been treated here by the tapering-off method, and all have said they suffered less, and had recuperated more quickly from the hyoscine than from any treatment ever tried.

My personal experience in treating morphinism is not very large, for, although my observation covers a period of 15 years, the number of alcoholic and narcotic habitués admitted into a general hospital for the insane is small; yet each year we treat a few. Prior to 1900 all were treated by gradually reducing the dose, and some are still treated in this way, but we have given 12 the hyoscine treatment.

While my success with the twelve cases has been most satisfactory, I would hesitate to recommend this treatment to the profession, were I not backed by men of much larger experience.

Dr. C. C. Stockard, of Atlanta, told me that he had treated over 300, and Dr. Geo. E. Petty, of Memphis, who is a pioneer in this method of treatment, claims that he has treated about 500 with the most gratifying results. I know both of these physicians personally, and have every reason to accept what they say as facts.

To give a clearer idea of this method of treatment, I shall report two cases without comment. These notes were made by my assistant, Dr. T. L. Dobson, during the treatment, and they are given as his observations.

CASE I.—R. C., male, age 22, general condition good. Lungs and heart normal; pulse 75. Has been taking morphine for several years, and now takes about 15 grains each day by the mouth. Second admission here, having been treated by gradually reducing the dose, but relapsed in a short time. Admitted July 10, 1900. Kept him under observation two days, then gave calomel at bed-time, following it the next morning with a saline.

July 12, gave usual morning dose of morphine, and began hyoscine at 2 P. M., using 1-200 grain.

2.15 P. M.; 1-200 gr. hyoscine.

4.30; 1-150 gr. Patient has been sleeping for two hours. Pulse, 60. Face very red, but doing well.

6.30; 1-100 gr. Still sleeping. On arousing called for his supper.

8.30; 1-100 gr. Sleeping, pulse 52. Complains of feeling very warm. Has not called for evening dose of morphine.

10.30; 1-100 gr. Not sleeping. Pulse 72. Complains of being cold. Speech thick and cannot articulate well. Talkative and partly delirious.

July 13, 1 A. M.; 1-100 gr. Doing well, but not sleeping and delirious.

3 A. M.; 1-100 gr. Condition unchanged. Pulse 72.

5 A. M.; 1-100 gr.

7.30 A. M.; 1-100 gr. H. Pulse 72. Says he does not want morphine. Bowels not moved, kidneys acting freely. Would not eat breakfast. Delirious and seeing bugs, but has sane moments.

10 A. M.; 1-100 gr. H. Has delusions, but is doing well and says he has no thought of morphine.

12 M.; 1-100 gr. Condition unchanged, except pulse is slightly intermittent. Delirious and hard to keep in bed.

1 P. M.; 1-30 gr. strychnine nitrate, and glass of milk punch. Took no dinner except milk. Complains of headache. Does not want morphine.

2.30; 1-150 gr. hyoscine. Pulse 82. Still delirious. Mouth and throat dry. Ordered mouth washed out with cold water every few minutes.

4.30; 1-100 gr. H. and 1-30 gr. strych. Pulse 90. Delirious and very nervous, picking at bed-clothes, sees bugs, and is

afraid of the hypodermic needle. Took half an ounce of whiskey.

6.30 P. M.; 1-200 gr. H. Temp. 100. Pulse 78. Very restless, seeing bugs, but does not want morphine.

10.30 P. M.; 1-250 gr. hyoscine and 1-30 gr. strych. Temp. 100°. Pulse 78. Seems more rational, but very restless and tossing about the bed. Takes whiskey every four hours, but refused morphine.

July 14, 8 A. M.; Temp. 100°. Pulse 84 and regular. Has had no hyoscine since last night at 10.30. Patient doing well and sitting up. Says he has no desire for morphine, but is nervous, and 15 grains of bromide of soda were given. He ate a little breakfast.

6 P. M.; Temp. 99.4°. Pulse regular. Feels perfectly well, and has no desire for morphine. Slept about two hours. Ate a good dinner, and has had lemonade freely. Kidneys acting freely. Called for bath. Prescribed a tonic of tinct. of nux vomica and tinct. cinchona comp.

This ended the treatment, and the patient was discharged in a few days apparently quite well.

Case 2.—E. W. Admitted August 4, 1900. Age 30. Condition bad; thin, anæmic and constipated. Heart weak, pulse 80. Asthmatic, and breathing difficult at times. Has taken morphine for asthma for several years, now taking about 30 grains morphine and as much cocaine as possible during the day. Has been treated several times by the gradual reduction method, as well as in a Keeley institute. Is opposed to the hyoscine treatment. After keeping him under observation for two days, and allowing him all the morphine he wanted, the special treatment was commenced after unloading the bowels.

August 7, 8 A. M.; 3 grs. morphine.

11.15 A. M.; 1-200 gr. hyoscine. Temp. 98°. Pulse 96. The quick pulse due to excitement incident to taking the hyoscine.

11.30 A. M.; 1-200 gr. hyoscine with 1-30 gr. strych. nitrate. Feeling weak, and complains of feeling very hungry.

12.45 P. M.; Sleeping soundly, but roused up and drank three cups of sweet milk and ate three small cakes. Does not call for drugs; says he is comfortable, but very weak.

2.00 P. M.; 1-200 gr. hyoscine. Was still sleeping; says he is badly drugged. Pulse 72, full and strong. Wants to eat.

4 P. M.; 1-150 gr. H. and 1-30 gr. strych. Sleeping when called. Ate a little. Begged for morphine and cocaine. Has begun to see bugs.

6 P. M.; 1-125 gr. H. Still begging for morphine and cocaine. Not well under the influence of the drug, but sees bugs, and says he cannot think or talk. Pulse 90.

8 P. M.; 1-125 gr. H. and 1-30 gr. strych. nitr. Still delirious, and sees bugs. Does not call for drugs. Had two eggs, some toast and sweet milk for supper. Pulse 66.

10 P. M.; 1-100 gr. H. Not so delirious, but sees bugs. Begs for morphine and cocaine. Has not slept any since 4 o'clock. Pulse 72.

12 P. M.; 1-100 gr. H. and 1-30 gr. strych. Doing very well, but still delirious and not sleeping. Sometimes calls for his drugs.

August 8, 2 A. M.; 1-100 gr. hyoscine. Still buggy.

4 A. M.; 1-100 gr. H. and 1-30 gr. strych. nitr. Still buggy.

7 A. M.; 1-100 gr. H. Pulse 60. Kidneys and bowels acting. Had milk-toast for breakfast. Still delirious and occasionally calling for morphine and cocaine. No evidence of suffering.

9 A. M.; 1-100 gr. H. 1-30 gr. strych. Pulse 66. Condition very good. Drank half a glass of milk punch, whiskey nauseating him. Delirious, but not calling for drugs as much. Sees bugs of all kinds.

11 A. M.; 1-100 gr. Pulse 60, good and strong. Still very delirious, and seeing more bugs than ever. Not calling for morphine.

2 P. M.; 1-200 gr. H. and 1-30 gr. strych. Thoroughly under the influence of the drug. Delirious and catching at bugs. Ate but little dinner, and vomited the few mouthfuls taken. Pulse 60.

4 P. M.; 1-200 gr. hyoscine. Very delirious, but called for drugs. Pulse 72, full and strong.

6 P. M.; 1-125 gr. H. and 1-30 gr. strych. Still wanting drugs not so much morphine, but "any old drug." Condition good. Pulse 66.

8 P. M.; 1-150 gr. hyoscine.

10.30 P. M.; 1-200 gr. H. and 1-30 gr. strych.

2.30 A. M.; 1-30 gr. strych.

August 9, 7 A. M.; 1-30 gr. strych. nitrate. Says he feels very badly. Called for drugs, but delirious and hardly knows what he wants. Vomited large quantity of bile after drinking some water. Retained lemonade.

10.30 A. M.; 1-30 gr. strychnine. Still nauseated and vomiting everything taken. Weak and condition bad. Gave 2 grs. calomel and soda.

1 P. M.; 1-4 gr. morphine to quiet stomach. Bowels acted at 1 o'clock, but still vomiting.

2 P. M.; 1-30 gr. strych. sulphate. Not so much nausea, but does not retain anything as yet. Feels some better.

4.30; Eating a peach and feels much better.

6. P. M.; 1-30 gr. strych. nitrate. Much improved, ate some supper and feeling very well. No nausea.

10 P. M.; 1-30 gr. strych. Doing well.

August 10, 2 A. M.; 1-30 gr. strych nitrate.

Aug. 10, 8 A. M.; Doing well. Gave tonic of tinct. cinchona comp., elix. gentian and chloride of iron and strychnine.

Aug. 11; Doing well. Does not want morphine or cocaine.

Aug. 17; Improving every day, and from this time made a good recovery, and never called for morphine. Sleeping well.

STATUS EPILEPTICUS: A CLINICAL AND PATHOLOGICAL STUDY IN EPILEPSY.

By L. PIERCE CLARK, M. D., New York, and THOMAS P. PROUT, M. D., New York.

(*Continued from page 306.*)

CONVULSIVE STAGE IN DETAIL.

The paroxysms of the convulsive stage of grand-mal status remain of the convulsive type throughout, or at least until exhaustion is extreme. Although status composed of "absences" attacks of vertigo, or simple losses of consciousness are very rarely fatal, this statement does not apply to the psychic attacks seen at the end of a grand-mal status in which the psychic character is due to the state of cerebral exhaustion—a condition in which it is not possible for the fatigued centers to respond in any other manner. As the convulsive stage advances in those cases which have an order of muscular invasion, the rapidity of involvement is delayed and the order becomes less definite; the whole cycle of an attack is also shortened in point of duration. Frequently, in the latter part of a convulsive stage of an unfavorable case, the first muscles to be involved are the only ones found to participate in the convulsions presenting imperfect or aborted attacks. This is especially true if convulsions begin about the eyes, mouth, or in the hand. Those cerebral centers which preside over most complex and arbitrary muscular movements are least exhausted, and consequently enter last into the general cerebral exhaustion paralysis, the terminal state of fatal status. Just as we see in serial epilepsy or status a more or less complete exhaustion paralysis in an extremity which was formerly but partially paralyzed from a cerebral lesion, so in fatal status we have an analogous general exhaustion. In other words, from a dynamic point of view, the status is a profligate expenditure of cerebral energy to that point or level at which a stimulus to

cerebral cells ceases to produce the phenomena of convulsions, there being a death paralysis. This fact was well shown in one of our cases of left infantile hemiplegia in which at the beginning of the status period the left side was first and most convulsed, but as it ultimately became paralyzed to the non-stimulative level, the right side was forced to continue the exhibition of cortical discharges in convulsions until finally it in turn became functionally reduced to a more or less paralytic state. This is not seen solely in hemiplegic epileptics but occurs invariably in idiopathic epilepsy, that masquerades as Jacksonian or partial epilepsy. The study of the convulsive stage of status is therefore really a pathological study of cerebral exhaustion. Near the end of the convulsive stage of status the paroxysms may be so slight and frequent that attacks pass unnoticed unless the observer is closely watching the case; indeed, a general choreiform tremor usually terminates fatal status in the convulsive stage.

Seizures may occur from 50 to 200 times per day; six of the 38 patients of this study had 50 to 100 attacks in 24 hours, while six more of the study had 100 to 200 attacks in the same period of time. One patient had 542 attacks in 48 hours, being one of the idiopathics having a definite order of muscular invasion. One of our cases, a hemiplegic epileptic, had frequently as many as 75 attacks in one day without being prostrated in bed, thus exemplifying in a striking manner that the single, serial or status period of such cases is not to be classed with grand-mal status of fulminant onset. The highest record of grand-mal attacks for any one case is probably held by one of Leroy's patients, who had 488 attacks in 24 hours; he had one thousand attacks in three days, which ended fatally. In contrast to the frequency of grand mal in this fatal case of status may be cited the psychic attacks of one of our patients, who had 755 "absences" or simple losses of consciousness in twelve hours. She was not at all prostrated from the series. Within one week another period of 500 attacks occurred. Some American writers have recently alluded to the extraordinary number of attacks which have taken place within a period of several weeks. Thus Parsons reports 1400 attacks in four weeks; others 2000 and 3000 in similar periods. It is needless to emphasize the fact that such interest-

ing statistics belong to epilepsy proper and not to a single status period, as status does not extend over such long periods of time.

The duration of the convulsive stage of status is very variable; it may last only three or four hours, or it may extend over a period of several days. The data upon which is based this paper shows one case at least in which convulsions persisted for over one week. As a general rule the convulsive stage lasts from a few hours (8 to 10) to three or four days. It forms about one-third to one-half of the whole status period.

STUPOROUS OR COMATOSE STAGE OF STATUS.

The stuporous stage of status is largely the product of the convulsive stage; usually the extent and degree of the coma is in direct proportion to the severity of the convulsive stage. Exceptions, however, are not very uncommon, but an alteration of the ratio is usually due to either a peculiarity of the convulsions or an interposition of complications of the stuporous stage. The stuporous stage, as such, exists for varying periods; but the severity of the status *per se* is not to be measured by the length or depth of the coma, as there are so very many modifying influences, the rallying power of the individual cases forming only one of the uncertain factors. Usually within a few hours after the convulsions have ceased the patient presents signs of rallying; there is marked improvement in the exhaustion symptoms, fever, pulse, and respiratory frequency. Complications of a varied sort often obscure the termination of this stage. To separate the favorable from the unfavorable signs in the face of confusing complications is always difficult. Edes has reported a case in which there was postponed exhaustion in which the stuporous stage appeared to return after recovery seemed assured. The patient suddenly collapsed; pulse, temperature and respiration ran high; the nates sloughed and death occurred after an apparently complete recovery from the stuporous stage. No autopsy was held. While such anomalous cases of status are not unknown in the experience of many observers, usually it is found that the autopsy clears the picture wonderfully. The post-mortem findings generally disclose a new complication which accounts for the renewed symptoms being the forerunners of a fatal termination of the case. The complications of status

come on suddenly and their occurrence, which under ordinary circumstances would be of little moment, aggravates in a truly alarming manner the condition of physical and mental exhaustion. If recovery is to take place the patient may become partly conscious and remain in an automatic state for several hours, but more frequently a mild delirium ensues which may deepen in intensity to active delirium. The occurrence of the latter state is particularly unfortunate, as death often occurs suddenly in a large percentage of post-status manias. The milder grade of mental reduction in the status is a noisy delirium in which hallucinations of hearing and sight play a prominent role. The patient may remain in this state being even mildly confused and delusional for days, weeks or several months before complete recovery obtains. The permanent insanities, from which many epileptics are said to suffer after status, are chargeable in greater part to the epilepsy proper rather than to the status. Mental alienation from status is usually an acute exhaustion psychosis tending toward recovery or at least toward the pre-status mental condition.

As a general rule the *duration* of a moderately severe type of status is not less than 30 hours. In the majority of cases it is found to be between two and nine days. In 130 cases, including those in this paper, 60.5 per cent were within these limits, 29 per cent were below the minimum, and 10.5 per cent exceeded the nine days. One case of this study ran one status period of 12 days which ended in recovery, yet the patient died finally in a status which was apparently less severe than former periods and lasted but 18 hours. The case was nevertheless an hemiplegic epilepsy which usually run a longer period of status than the grand-mal idiopathic type. However, if it is sometimes difficult to say just where a status begins, it is doubly difficult to mark its period of exact termination in every case. Most authors agree that the return of consciousness after the stuporous stage determines the end of a status period. If either stage is curtailed in status, it is that of stupor. About one-half of the fatal cases have no real stuporous stage, there being but a few moments' cessation of the attacks before the death agony.

TRANSITORY HEMIPLEGIA IN STATUS EPILEPTICUS.

As might be expected, we have many disorders of motility after the status. For the most part they have their origin in the general exhaustion resulting from the extraordinary expenditure of energy. Temporary exhaustive paralytic disorders may manifest themselves in defective special sense functions, in anesthesias, paresthesias, tremors, incoordination of bodily movements in locomotion, which at times may only amount to mere awkwardness; or there may be purposeless, aimless movements, choreiform or athetoid in character, and the like. These temporary paralytic states are dependent upon cerebral exhaustion and not primarily upon a defect in function of the muscular apparatus, so recently engaged in the paroxysmal display. We have already alluded to the fact that some authorities still hold local transitory paralysis to be pathognomonic of the status condition and therefore believe that the underlying causative lesion of the state is an apoplexy. While a sort of status not infrequently occurs in cerebral apoplexy yet it differs in many respects from true status epilepticus, and the autopsy findings recorded in the past and those to be given later conclusively prove the status a non-apoplectic affection. Transient exhaustion paralysis is always a feature of status unilateralis of organic epilepsies, i. e. those epilepsies having for their primary cause an infantile cerebral palsy. Jackson has long held that the coma and temporary general relaxation which follow ordinary attacks of grand mal are analogous to the local paresis or paralysis which is present after the local or partial paroxysms. It is a well-recognized fact that the status epilepticus, which is the very climax or acme of epilepsy, is followed by precisely the same sort of transitory general palsy as obtains locally after monospastic seizures of focal epilepsy; but the general exhaustion is not to be detected inasmuch as it is covered by coma and because the recuperative power of fatigued ganglion cell and non-medullated nerve-fiber is extremely rapid. However, to show that transitory paralysis may occur without a status period, Gowers has studied the phenomenon extensively and yet has never seen but few cases of true status. Our experience accords with that of Nothnagel's (Epilepsie, Ziemssen's Handbuch, 1875), who holds that temporary hemiplegia does not invariably

occur in status, but that it ensues if the status is composed of paroxysms partial in character or with a focal maximum of intensity. Lorenz collected complete notes of 38 cases and found transitory hemiplegia mentioned in but four (Leroy two; Hertz one; Bourneville one). Edes cites the occurrence of transitory hemiplegia and disbelieves in its being due to a gross lesion rather than to exhaustion. Voisin speaks of a case of status in which the paralysis appeared and disappeared several times during one status period. This is not an uncommon clinical observation; we have seen several such cases. The paralysis in spasmodic infantile hemiplegias always deepens in the status, for two very obvious reasons; the cerebral centers presiding over the paralytic side have already been weakened and damaged by the primary cerebral lesion and its weakness and instability cause it to be first and most involved in the cortical discharges.

As has so often been stated in this thesis and elsewhere, the early hemiplegia frequently goes unrecognized until a status period or several epileptic attacks cause the paralytic state to become exaggerated and therefore obvious. Again, exhaustive paralytic phenomena may occur in idiopathic epileptics. But in studying all such phenomena the general law of Jackson holds good, that paralysis follows in parts first and most involved in convulsions. This fact is often overlooked by investigators. Thus Lowenfeld, Haidenhain, Gowers and other inhibitionists, fail to see that temporary paralysis is a result of a cerebral exhaustion and is not always to be measured according to the varying severity of the antecedent convulsive phenomena. Postmortem findings in the temporary exhaustion paralysis cases do not disclose any hemorrhagic foci in the cerebral centers presiding over paralyzed parts, neither are they found in the hemiplegic epilepsies. Several cases in this study of both kinds of epilepsy prove this point. We are therefore forced to interpret these transient pareses in status, as well as in epilepsy proper, as being purely states of cerebral exhaustion. The nervous centers are unequally charged and the discharges from the different centers are not the same, and as a result certain areas in the cerebral cortex are left paralyzed, while others are but slightly weakened. It is barely possible that there may be a few

disguised infantile hemiplegic epileptics who may have general grand-mal attacks with local transitory hemiplegia, and that in this way the occurrence of exhaustion paralysis in the idiopathics is to be accounted for. But from accurate clinical data we know that this is hardly probable, for all infantile hemiplegic epileptics have their paroxysms most severely in the hemiplegic parts, the parts in which the first paroxysms of a beginning epilepsy originate.

The Reflexes in Status.—The deep and superficial reflexes are lost as soon as the comatose stage in the convulsive period becomes continuous between paroxysms and invariably remain absent throughout the stuporous stage. They can again be elicited just before consciousness is regained; their return being often the first indication that consciousness will reappear. Babinski's sign is always most pronounced after status unilateralis of infantile hemiplegic cases and is present in a lesser degree in the majority of cases showing the transitory paralysis independent of a previous cerebral lesion. The reflexes remain absent longest in the parts most convulsed and later become most exaggerated in them. This also applies to Babinski's sign. As this phenomena appears to rest upon a destruction, irritation or possibly exhaustion of the pyramidal tract, its alteration in status is of considerable moment from a diagnostic view-point. The deep reflexes after status composed of grand-mal attacks remain uniformly exaggerated for weeks or months, and the time often serves to measure the degree of exhaustion from which the cerebral cortex has suffered. This fact was first noticed in the status of the hemiplegic epileptic with hemispastic or monospastic seizures, and goes to prove that the pathogenesis of the cerebral discharges in organic and the so-called symptomatic epilepsies, as well as the resultant local and general paralyses from both clinical states, are identical, the difference being one of degree only. As our knowledge of the nervous system advances, the homologies in all the now widely separated convulsive disorders must prove more and more that there is an essential unity in their pathogenesis throughout.

Mouth, Pharynx and Nares.—As status develops the saliva ceases to form on the lips or in the mouth owing to glandular inactivity. The lips, cheeks and tongue may be fissured and

bleed easily. In addition to showing fissures, the tongue, lips and cheeks may also become a mass of sores caused by the patient biting them in the convulsions. Herpes of the lips and face form frequently and are occasionally seen on other parts of the body. Sordes in thick crusts collect on the teeth and lips and the breath is extremely offensive. As soon as unconsciousness is continuous the patient's throat is filled with mucus and this is kept in noisy agitation by the respiration. In all severe status there is gradual pharyngeal paralysis from the first few convulsions, which renders the taking of food and drink both difficult and dangerous. This phase of exhaustion paralysis is not fatal in itself but often remains more or less permanent even after convalescence is well inaugurated. The posterior nares become clogged with mucus in the convulsive stage, while in the stupor period the anterior nares are frequently excoriated and fissured. Epistaxis may repeatedly take place in the latter part of the stuporous stage in a most distressingly persistent manner. More or less severe and prolonged hemoptysis may also occur in status, as has been noted after isolated paroxysms of epilepsy proper. The occurrence of hemorrhages from the lungs warrants a bad prognosis.

Pupils and Eyes.—The pupils may undergo a variety of changes. In the early convulsive period they are usually dilated during the convulsions and contract to the normal after paroxysms, but gradually as the stupor deepens they dilate and remain insensible to light throughout the remainder of the status. Generally they are dilated equally, but when this is not so the convulsion on the side of the more mydriatic pupil is more intense and persistent than on the other. In exceptional cases the pupils may be found of the pin-head type, similar to the pupil of opium narcosis. Not infrequently in the status the pupils become elliptical in shape or have a wavy outline at the onset of the convulsions composed of general tremor. They may also change in size with the inspiration and expiration, increasing with the former and decreasing with the latter. An explanation of many of the pupillary changes in status rests upon asphyxiation and exhaustion. The eyeballs in the later stages of status keep up a more or less persistent lateral nystagmus, but occasionally the movement is rotatory; more frequently a combination of both

movements is seen. When not in motion the eyeballs are rolled upwards and internally. The conjunctivæ are intensely injected and frequently show hemorrhagic infiltration, either in one large spot at the outer or inner third of the orbit or in several small scattered areas spread more or less evenly over their whole extent. If hemorrhages occur in both conjunctivæ they are usually symmetrical in degree and distribution in both eyes. Trophic ulcers may form on either or both corneæ; more frequently the condition occurs in those status cases in which exhaustion-paralysis obtains and is then found on the side most convulsed in the status.

The Skin in Status.—The condition of the skin, which is covered with cold clammy sweat in the later stages of status, is in striking contrast with the real heat of the body shown by the axillary or rectal temperature. In regard to cutaneous rashes, Lorenz has noticed an erythematous roseola in one of his cases. Some form of an erythema over the trunk, extremity or face, or over the entire body is far from uncommon. Usually such skin rashes appear on the cheeks or forehead. Urticaria is often noticed; it may be of the common or bullous type or of the giant variety, and may shade imperceptibly into a condition identical with or analogous to acute circumscribed œdema. At times the giant urticaria form of tropho-neurosis of the skin has been known to herald attacks in series, and even the status itself.

The *tâche cérébrale* of Trousseau, the meningitic streak of Bourneville, or better the dermographism of Feré and Lamy is always an accompanying symptom of severe status. As is to be expected from the real nature of the phenomena its intensity and persistence in status depends upon the degree of exhaustion of the vaso-motor apparatus which, as we before observed, is the real index to the general vascular exhaustion. The condition is no longer held to be pathognomonic of any inflammatory state of the brain, it is probably sympathetic in origin and is not necessarily connected with any peculiar cerebral state other than those inducing exhaustion. The white and blue œdema of Sydenham and Charcot are said to be occasionally accompaniments of the status epilepticus, but as such conditions are of particularly common occurrence in the status hystericus, one is

led to doubt the diagnosis of the form of status in which these peculiar œdemas are said to be found. We have never seen the condition in status epilepticus, although it has occurred in two cases of status hystericus under our observation.

The frequency in which subcutaneous hemorrhages occur in grand-mal status is well known. They are probably present, to some extent in every severe case. Just as in epilepsy proper so in status they occur; first, on some part of the face, next on the neck or chest and finally on the posterior and external aspects of the trunk and extremities. The peculiarities in their distribution are many and interesting. The degree is largely a matter depending upon the intensity and length of individual paroxysms of the status and their influence on the vascular apparatus. Minute punctiform hemorrhages may be found over the entire skin surface in exceptional cases of the mildest petit mal of epilepsy proper, or, we may encounter large irregular blotches after one grand-mal attack. The mucous membranes and internal organs present similar capillary hemorrhages, which are also probably due to the same cause, namely, arterial spasm. The blood pressure is enormously increased by the muscular spasm of a seizure and has been carefully investigated by Feré and Norwatsky and Arndt in isolated attacks and in status epilepticus. The hemorrhages may occur in the status of psychic seizures, but in all such cases the blood pressure has been found as much increased as in the grand mal variety, although severe convulsions of the skeletal muscles may have been absent. Undoubtedly individual idiosyncrasy and the state of nutrition and exhaustion of the vascular apparatus count for as much here as they do in the so-called bleeders or those suffering from purpura hemorrhagica. Then, too, certain kinds of epileptic attacks involve the peripheral circulatory apparatus. The importance of the presence of vaso-motor spasm had been much over-estimated in explaining so-called vaso-motor epilepsy and epilepsy in general. The subcutaneous hemorrhages usually disappear entirely in a few days if the status patient convalesces. Exceptionally it has been known to leave a permanent pigmentation or bronzing of the skin after status, but this is a rare termination.

Trophic Disorders.—The trophic disorders of status are very

important. Bodily wasting is often extreme; patients have been known to lose twenty pounds as a result of but eight hours of status. The usual loss of weight is about three or four pounds for each day of continuous convulsions, varying from this general rule according to the severity of the status, the amount of food taken and the individual capacity of resistance to fatigue. It may be said that there are cases of status equivalents composed of stupor in which extreme emaciation occurs as the most prominent and alarming symptom. It is still mooted whether sudden and extraordinary loss of bodily weight in an epileptic could ever be classed as a status equivalent, as has been urged in epilepsy proper by Kovalevsky. However, the relationship between different psychic states and nutrition is not at all understood, as is evidenced by the confusion still existing in regard to the tropho-neuroses in general. It is entirely conservative to state that the rapid emaciation and death that occur in some epileptics when no adequate cause is demonstrable post mortem may form a nutritional state analogous to status. Even now it would be as tenable as the cause of death in many epileptics ascribed to "epileptic dyscrasia." In many of such cases the state of the ductless glands, such as the thymus, cannot be urged as sufficiently abnormal in structure or function to cause death in epileptics.

The most marked single evidence of the tropho-neuroses is the persistent forming of bed-sores in all severe or prolonged status. The sloughing is similar in character and location to that seen in general paresis and the acute psychoses where rapid mental and physical dissolution takes place. Sloughing of the nates occurs on the side most convulsed and therefore most exhausted, or in status unilateralis where the seizures are confined entirely to one side, as is usually the case in the status of general paralysis. Bed-sores are not entirely confined to the nates, as sloughs frequently form over the scapular prominences, on the heels, or on any dependent portion of the body where persistent pressure is made. The soiling of the clothing by feces and urine adds materially to the sloughing.

The skin and its appendages also suffer from nutritional defect as a result of the status. More or less complete desquamation may follow in the wake of a convalescent state. The growth of

the hair and nails may also be disturbed; the hair may fall out, split, or break off near the scalp, while the nails frequently become ridged, both longitudinally and laterally; they are usually brittle and flecked for some weeks following status.

The Bodily Secretions and Excretions in Status.—The urine is generally voided unconsciously at the outset or as soon as the inter-paroxysmal coma is continuous. It is scanty, highly colored and loaded with phosphates. In all severe status it usually contains traces of albumin and blood. The amount and constancy with which albumin is found can be fixed by no general rule. At times it may be in great excess; again it may be entirely absent even in severe status, but generally there is at least a trace of albumin. Much experience forces one to the conclusion that the amount of albumin is largely dependent upon the degree of asphyxia and of renal insufficiency. In the later stages of fatal status the urine may contain both hyalin and granular casts, and urates appear in large quantities. The toxicity of the urine in severe status is much increased.

The feces are less frequently voided voluntarily than the urine, and the fecal matter in advanced status may present much undigested food. While constipation is the general rule in status, a diarrhea may be present from the start; but this latter is of quite uncommon occurrence. In such cases, in addition to the involvement of the muscles of the abdomen, there is usually an extension of the epileptic discharge to the coats of the intestinal walls which causes increased peristalsis and expulsion of their contents by the rectum, as shown clinically and experimentally by Ossipow. In most fatal status cases, however, obstinate constipation obtains.

The sweat and blood in status undergo marked changes proportionate to the relative severity of status. That profuse perspiration usually attends severe status is well known. Its occurrence in the first stage is due to over-activity of the sweat glands, provoked in greater part by the pathological heat production from the paroxysmal activity and occurs secondarily by exhaustion. The patient's bed-clothing is usually saturated with sweat in a few seconds. The sweating is usually general, even when the convulsions are localized. The toxicity of the sweat in the different status periods has been variously estimated

by different investigators. Usually it increases with the length of the status and is directly dependent upon the quantity of waste products eliminated through the skin. This statement holds true for the blood and urine also. The patient's loss of bodily weight in status, which may amount to from 5 to 30 pounds, is due in great part to the elimination of the liquids of the body by sweating. At first, the sweat is like that of an individual undergoing great muscular exertions, but in the later stages it is very clammy, and its solid constituents increase. But few investigations have been carefully made upon the blood changes in status; the leucocytosis has an important clinical import. Some authorities hold that the blood contains primarily a toxic agent which precipitates the status; but such a theory is entirely without substantiation at present.

Fever Curve.—The elevation of the bodily temperature in status usually begins after the first severe convulsion, amounting to from one-half to one degree; or it may be delayed until several seizures have occurred; usually there is a marked rise after six or seven. In the status composed of partial paroxysms or those with a definite order of muscular involvement, it is not uncommon to see the temperature remain normal after 30 or 40 paroxysms have occurred; even in the status composed of the incessant clonic, monospastic or hemispastic seizures the temperature usually rises very slowly and subsides quickly at the cessation of the paroxysms. As a general rule, other things being equal, it may be stated that there exists a direct ratio between the number and severity of the convulsions and the resultant fever in the status. But this rule holds good for the idiopathic grand-mal status only, and as complications often occur, the disparity of this ratio is by no means uncommon. Its constancy at the onset of status holds sufficiently true as to warrant us in stating that there is a cause and effect relationship between the paroxysms and its fever curve. The occurrence of fever in the status equivalents are dependent upon other laws, the nature of which we do not understand. Elevation of temperature is invariable in the status of grand mal. The degree of elevation and the state of pulse and respiratory frequency constitute the best index of the severity of any particular status period.

Lorenz believes that there are two maximum points in the fever curve; one for the convulsive and one for the stuporous stage. After a careful review of all cases found in the literature (about 120 including those of this study), the two curves are found in but 30 per cent. This rule, therefore, is evidently far from being general. A decided advance of temperature in the period of stupor should cause us to expect complications, and therefore an unfavorable termination. The temperature generally rises to the maximum more rapidly than it subsides, even in the most favorable cases. Occasionally the fever ceases by crisis. That the temperature may go up to 107, 108 or even 109 is not unknown; indeed, the curve may continue to rise after death, as is often seen in fatal sunstroke. It may be several hours before the normal is reached. Bourneville has reported this phenomenon in several fatal status cases, and we have seen it in three instances. This pathological formation of body temperature is generally seen in epileptics dying in the midst of convulsions, but the contrary does not hold true; we have looked diligently for its occurrence in fatal cases dying in the stuporous stage, but have not found it except in one case. However, it is not to be found in all patients dying in the convulsive stage, but chiefly in those who die in acute status, the duration of which has extended over a few hours. Finally, it must be said that fever is not invariably present even in acute status. In those fatal cases of status where post-mortem rigidity is absent for 24 to 46 hours after death, if tested, a rise of temperature in some degree would probably be found. The phenomenon is a very interesting one, showing that many of the vital processes in the cell and even in entire organs of an organism, may continue their activity for hours after the complete suspension of respiration and circulation, which are the ordinary signs of death. Until our knowledge of the chemistry and physiology of the individual cell is more definite, and the specialized functions of cells and organs are better understood, we cannot hope for a rational explanation of this morbid heat phenomenon in fatal status. Authorities still differ widely even as to the approximate cause of the fever curve in any status case. Certain investigators report status periods in which there is but little or no fever curve following severe convulsions, and maintain that such cases destroy

the theory of relationship supposed to exist between the muscular convulsions and the fever state. Such cases may be found, but if they are studied closely some anomaly of the convulsions will be found to account for the exception. On the other hand, it is much more difficult to explain the actual occurrence of extreme fever and death with but slight convulsions. Here a warning may not be misplaced in cautioning the physician not to judge of the severity of an epilepsy by the number and severity of grand-mal convulsions; some of the most disastrous effects of the epilepsies, physically and mentally, are manifested through psychical symptoms only.

Even if the presence of meningitis should be granted, which Bourneville holds to be the underlying pathology of the stuporous stage, it would not answer the inquiry as to the fever curve; neither would pneumonia or pleural complications solve the problem, for the cerebral as well as the pulmonary conditions may exist and yet the fever curve reach normal in the stuporous stage. Finally, there are innumerable instances in which persistent high temperature has been recorded and no complications have been found after death to account for the temperature rise. Nothnagel still thinks the fever of the convulsive stage is similar to the temperature phenomena observed in certain diseases of the central nervous system during the death agony; that the unexplained sudden rise of temperature in general paresis as well as the febrile movement which sometimes accompanies fright in children may be akin to it; in other words, these cerebral disturbances affect the heat centers. Witkowski counts the temperature rise purely psychical, the fever being a sort of equivalent or congener of the delirium, sleep, stupor, &c. He holds that the fever subsides when the convulsion ceases. This is generally true, but the fact that the opposite does not obtain, destroys the whole theory. If there were a heat center in the cortex, as Eulenberg and Landois taught, it would not be difficult to explain fever in an epilepsy of cortical origin, but recent experiments have exploded the possibility of the existence of such centers. However, the fever of status is not more mysterious than the periodic loss of consciousness independent of and following paroxysms of epilepsy proper.

The fever resulting from the first few attacks can be directly

ascribed to the pathological excess of heat liberation from the convulsions; the continued rise even after the paroxysms tend to lessen in severity, may be accounted for by the presence of the waste products or toxins in the circulation which are well known to produce an experimental reaction when injected into the healthy animal. The athlete's fever is a sort of physiological prototype of the early convulsive state of the status, and is susceptible of the same interpretations. Nor is this all. In most status cases all secretory activity is at a minimum because the glandular structures participate in the general exhaustion. The cessation of excretion of the kidneys alone adds materially to secondary intoxication, thus producing a sort of secondary uræmic fever. Such a condition is especially liable to occur in status, inasmuch as the vitality of the organism is incapable of resisting any sort of toxic agent. The toxicity resulting from a failure to excrete waste metabolism is quickly expressed in a febrile state in an epileptic previously exhausted. Hence we would expect the fever to subside as the organism recovers from exhaustion and overcomes the toxicity; this is exactly the clinical dissolution of the febrile movement. In every case of grand-mal status the fever decreases as the exhaustion subsides and physical and mental recuperation is brought about through food and rest. Moreover, the state of coma which is largely a psychical expression of exhaustion in status also lessens as exhaustion and fever subside. Undoubtedly there are many secondary histological changes of the cerebral cortex produced by the toxicity of the status itself. Again, persistent high fever in itself must be reckoned with in the post-mortem changes of status. Many of the meningeo-encephalopathic changes described by Bourneville have their origin in the secondary intoxication and its resultant fever. However, it is not yet possible for us to detail the exact rôle of the toxicity and the secondary fever of status, nor the part played for and against these changes by the different organs of the body in such a complicated problem as the status. That there are many modifying factors of the fever curve still unknown may be surmised by the number of exceptions and variations; any general rule governing the same, however broad and comprehensive it may be, would be too hazardous to formulate.

Pulse Curve.—A large part of the exhaustion from the status is borne by the vascular apparatus. The pulse-rate increases in frequency after each attack—which is the vascular index of exhaustion. Vaso-motor control is lost through a temporary paresis and the blood pressure in consequence is markedly lowered immediately after the paroxysm. In the early stages of status the blood pressure is increased three or four times that of the normal, owing in large part to the muscular spasm; but after the convulsive stage is well on, the arterial pressure may be below normal; the peripheral control being lost through paralysis of the vaso-constrictors. No organ shows the slighter grades of exhaustion more prominently than the peripheral vascular apparatus; even after single convulsions the paresis of the vaso-constrictors is often evident for hours and days, and in the chronic epileptics who have frequent seizures the condition of peripheral exhaustion becomes permanent and forms a large part of the epileptic facies. After a favorable termination of severe status the vaso-motor paresis often exists in some degree for weeks. The intermittent accretions of exhaustion following frequent paroxysms are borne by the heart for a longer or shorter period of time until it shows permanent signs of non-recuperative power between attacks; and a persistent acceleration results. Irregularity of force and rhythm are constantly found in the accelerated heart action. The pulse curve generally follows a course parallel to that of the fever curve through the early part of the convulsive stage; but variations are not infrequently found. The pulse curve depends upon the paroxysmal frequency and severity, and therefore its modifications in the course of the convulsive stage afford a more accurate index of the degree of exhaustion than those of the fever curve considered separately.

Respiratory Curve.—The exhaustion of the respiratory apparatus is the latest phase of status. The curve corresponds in the main with the other two, especially in the latter part of the convulsive stage, when death from exhaustion becomes imminent. The respiration in the later stages of status becomes irregular, shallow and very rapid. Cheyne-Stokes respiration is usually found in all severe status, but is far from being necessarily a fatal sign, as many patients have it and recover from status. When attacks are very frequent, sufficient oxygenation of the blood is

not possible and chronic asphyxiation obtains, with its resultant evils. The defective oxygenation favors the secondary intoxication of the organism, and the occurrence of albuminuria is generally proportionate to the degree and persistence of asphyxiation affecting the blood, excretion and secretion.

A sudden marked disturbance in the respiratory rhythm is often the only sign of lung complication, especially in the latter part of the stuporous stage. The most frequent of these complications are in the following order: hypostatic congestion, is almost invariably present in some degree; broncho-pneumonia —a more or less severe bronchitis is found in nearly every severe case; and lobar pneumonia, following the order of lobe involvement as in ordinary pneumonia. Pleuritis, other than that of the mild plastic type, is rare in status. It is particularly unfortunate that many of the grave symptoms of lung complications of the stuporous stage cannot be differentiated from the symptoms naturally seen in a patient from this period. The resultant symptoms from both are very variable in their manifestations.

Differential Diagnosis of Status Epilepticus.—A comparative study of the different status conditions belonging to such diseases as uræmia, apoplexy, general paresis, tetanus, hysteria, epilepsy, puerperal and infantile eclampsia, and the status of the different drug intoxications, has not yet been written and until this is done the differential diagnosis of status epilepticus must remain imperfectly presented.

However, when the history of genuine epilepsy is obtainable, the diagnosis as a rule is rendered comparatively easy by considering the presence or absence of the cardinal symptoms. In the absence of a history of epilepsy the convulsive period of status may be confounded with any of the diseases heretofore mentioned in this section. The urinary findings peculiar to the uræmic state are not observed in status epilepticus, although the presence of albumin in large quantities is common enough in the latter condition. Casts are almost never found in the status of epilepsy, except in those patients who finally die of Bright's disease. Again, the stuporous stage of status epilepticus may be confounded with the long category of comas, such as uræmia, apoplexy, alcohol, opium, etc. As a general rule the stuporous stage of status epilepticus is easy to differentiate from the coma of brain

or meningeal hemorrhage, if the fever, pulse-rate and respiratory changes of the former are carefully studied. Diabetic coma can be differentiated from status epilepticus because of the absence of fever and the presence of urinary findings in the former disease. Apoplectic coma can be distinguished from status by the presence of dilated and equal pupils in the latter affection; also by the infrequent, incomplete and transient hemiplegia seen in status. Epileptiform attacks of general paresis are identical with those seen in status epilepticus unilateralis, but the latter are as rare as the former are common and the history of the two affections leading to the status can hardly be confounded. The peculiar history and character of the spasm in tetanus is rarely seen in epilepsy, there being but three or four cases of status epilepticus of tetanoid character on record. The differential diagnosis of the rabietic status is only important in animals; in such, as veterinary science progresses, an astonishing number of animals will be found destroyed in the status epilepticus which has heretofore been supposed to be status rabieticus. The frequent absence of typical lesions of rabies will then be explicable on the ground of an error in diagnosis.

Puerperal eclampsia is differentiated from status epilepticus by noting the character of convulsions. Epileptic seizures are usually both tonic and clonic, while puerperal eclampsia is largely tonic and there is no tongue biting. By far the most important and greatest difficulty in differential diagnosis belongs to status hystericus and status epilepticus. While the disproportion between the frequency and severity of paroxysms and their resultant effect on the cardinal symptoms is the ranking differential test, the absence of profound coma and exhaustion appear to be the truest guide. Status hystericus may have some degree of temperature elevation, but usually the cardinal symptoms remain uninfluenced. Karplus has recently overthrown the differential pupil test, and the stigmata of hysteria are often absent or difficult to detect in the course of status hystericus, but return prominently and promptly as recovery obtains. There are not a few epileptics who have major hysteria also; in such, status hystericus or status epilepticus may occur. Again, the symptoms of hysteria and epilepsy may alternate in the development of a conglomerate status, but usually as the condition progresses the sta-

tus hystericus is fully and completely eliminated. Not infrequently idiopathic grand mal manifests itself in violent contortions, fury and passionate attitudes, which in many ways simulate major hysteria, but careful attention will almost always quickly dissipate the illusion, especially if the exhaustion and fever be noted. The opisthotonos of tetanoid status epilepticus is strikingly similar to the morbid exhibitionism of hysteria so characteristically illustrated in Richet's classic work upon the subject. However, a careful grouping of all the phenomena presented in any single case will usually determine the diagnosis between these by no means widely separated diseases.

Prognosis of Status.—The prognosis of status is necessarily grave, since it is recognized as the severest form of the epileptic state. A low temperature is not necessarily a favorable symptom. There have been numerous recoveries with 104° F., and 106 F.; even a temperature of 107° F. is not absolutely prognostic of death, as Lorenz holds. One of our patients had a temperature of 107.4° F. and recovered from that particular status period only to die some months later in another status associated with a much lower temperature. The individual resistance, the suddenness of onset, and the severity of the attack, as well as the fever curve, must enter into the prognosis. Extreme paralysis of the pharynx is an unfavorable symptom, but many patients have this symptom well marked and, nevertheless, recover. Often the paralysis in these cases is so marked that for several days after full convalescence, liquid food is taken with the greatest difficulty; there is often persistent and distressing nasal regurgitation. As heretofore noted, some degree of bulbar paralysis is present from the onset of all severe status.

The duration of the status is not an absolute factor in the prognosis, as Bourneville has held; patients may recover after nine, ten, or even twelve days' duration of the disease. One of our cases well illustrates this point; this patient finally died in a status that lasted only 18 hours. It is usually the status unilateralis or status composed of hemi- or monospasm that may be markedly prolonged. In proportion as the paroxysm of any status case varies from the idiopathic grand mal without order of invasion, the possibility of a fatal termination lessens. The shorter the interparoxysmal periods between the first few at-

tacks, the worse the prognosis; in such cases acute exhaustion is imminent, and but few remedies avail against this state. To be able to postpone the paroxysms as soon as the first fury of the onset occurs is the strategic point in treatment, hence the prognosis is based in large part upon the readiness with which the paroxysms yield to the emergency treatment. The extremes of age render the prognosis for recovery much poorer than in an epileptic of middle life. Women are supposed to stand status better than men, but the rule cannot be proven by data. The rapidity with which exhaustion deepens with the individual paroxysms and the history of the gravity of previous status periods, assist much in forming definite prognosis of the outcome of the individual case. The early presence of large quantities of albumin and blood in the urine coupled with great exhaustion, are very unfavorable symptoms. Finally, the early onset of continuous coma renders the prognosis very bad.

The early occurrence of complications in status portends a fatal termination. A sudden rise or fall in any one of the three cardinal curves (fever, pulse or respiration); a sudden cessation of convulsions without evidences of returning consciousness; or the marked alteration of any one of the cardinal symptoms without the others showing a corresponding change, are absolutely fatal signs; such a change almost always occurs from six to eight hours before death.

Before Lorenz collected the notes of 80 cases from different authors, the percentage of recovery was held by Nothnagal, Binswanger and Legrand du Saulle to be a little less than 50 per cent; Lorenz, however, found it to be about 45 per cent. In our study of 52 cases, 14 died while 28 recovered, which gives a mortality rate of 33⅓ per cent. It is but just to compute the mortality from status on the total number of status periods instead of basing our calculations upon the number of individual patients treated. If this is done, in our study of 100 status periods the mortality rate becomes 14 per cent—86 per cent of recovery. Many patients have a record of 3 or 4 status periods. One of ours had 7 before a fatal period occurred. No one factor enters the prognosis more prominently than the history of serial or status periods and their favorable response to sedative treatment.

In conclusion, the general rule for basing an unfavorable prognosis in status may be laid down when the gradual, deliberate,

step-like increase of the cardinal symptoms obtain and remain unmodified by sedatives.

Pathology.

The literature bearing upon the nature and pathology of epilepsy has increased enormously during the past fourteen years, and the views held by various writers have multiplied proportionately. The rapid change in our ideas regarding the nature of epilepsy is, however, evidence of positive advancement, and fluctuations of opinion in recent years are not so extreme as formerly. That epilepsy is due to a spasm of the arterioles; that it has its seat in a diseased condition of a so-called convulsive center; that it is due to a disease of the sympathetic nervous system; that its primary seat is in the medulla and basal ganglia; that sclerosis of the cornu ammonis is the essential lesion; that cerebral anæmia causes the convulsion; are theories which now find practically no advocates, to say nothing of other views long buried beneath the dust of many decades. Since the advancements of recent years in cerebral localization opinion has gradually centered upon the cerebral cortex as the primary seat of the nervous storm in epilepsy. However, some of the older theories have persisted in greater or lesser degree until quite recently. Gowers writing as late as 1893, felt constrained to controvert the expiring hypothesis of the origin of epilepsy in vaso-constriction and consequent cerebral anæmia, the evidence of which was based upon the existence of pallor at the outset of the fit, a very inconstant symptom.

It may be stated with certainty that the work of recent years has eliminated the medulla and basal ganglia as the primary seat of the nervous storm in epilepsy. A large share of the credit for this work must go to the experimental physiologists and pathologists and collaborating clinicians, a partial list of which includes the names of Hughlings Jackson, Ferrier, Horsley, Luciani, Unverricht, Munk and Schafer. As regards epilepsy in particular the writings of Hughlings Jackson during the past thirty years have reflected the newer facts in cerebral physiology. More than thirteen years ago this author expressed his belief in the cortical origin of epilepsy, an opinion foreshadowed in many of his previous writings. He further stated that genuine epi-

lepsy occurs, most probably, only in connection with the higher portions of the cerebral cortex. Christian, writing ten years ago, subscribed to many of the doctrines of Jackson, but suggested the localization in the anterior part of the cerebral hemispheres. He further expressed his belief in the essentially secondary character of the motor manifestations of epilepsy.

Among the first to describe a microscopic lesion in the cells of the cerebral cortex were Bevan Lewis and Van Gieson. The former found vacuolation of the cells of the second cortical layer in all of his cases, a point of some interest in connection with this paper, while the latter described neuroglia changes and others of an indefinite character in the cells of the cortex. Bevan Lewis' findings were demonstrated by means of a fresh method which, because of its unreliability, has since gone out of use. Juliusberger studied the changes in the ganglion cells of the cord in two status cases by means of the Nissl method. No positive changes aside from chromatolysis were demonstrated. Staining and displacement of the nucleus are mentioned. There was no study of the cortex. Collins described the microscopical findings in two cases of status. Changes in the blood vascular structure of the cortex were found which he thought gave rise to localized softened areas which were ultimately replaced by neuroglia tissue. Swelling and vacuolation of the protoplasm of the large pyramidal cells were also described. It is to be noted that both of these authors found changes of an acute exhaustive type in the cells examined, the more marked alterations being found by Collins in the cortex. Changes of this character have more recently been observed by Mott in both the brain and spinal cord in status. He regards the observed conditions as similar to those described by Goldscheider and Flatau, and Marinesco in hyperpyrexia, and believes he has evidence which points to a chemically altered lymph environment of the cell. These conditions were most marked in the brain cortex.

Another class of changes have been described by Roncoroni who found certain morphologic anomalies in the cortex of epileptics in general. These consisted in a decrease in the cells of the fourth cortical layer and the occurrence of an unusual number of nerve cells in the subcortical white matter. In this connection also Alzheimer describes a broadening of the first cortical layer

with great diminution in the number of cells of the second layer. These changes were accompanied by chromatolysis and gliosis, and a great diminution in the number of tangential fibers.

Among recent writers who regard the neuroglia hyperplasia in epilepsy as of especial importance are Chaslin and Feré. These writers lay special stress upon the gliosis in epilepsy and regard the Ammon's horn sclerosis, found in epilepsy, as a circumscribed manifestation of a wide-spread glial change. Changes in the nerve-cells are regarded as secondary. Bleuler agrees in the main with Chaslin and Feré. In twenty-six cases examined, this author found pronounced gliosis in the outer cortical layer in all. He concludes that the neuroglia changes are not proportional to the duration of the epilepsy, but correspond rather to the degree of mental involvement. He believes that the gliosis of epilepsy is distinct and can be differentiated from that of other conditions. Binswanger and Oppenheim recognize the importance of the gliosis of the cerebral cortex in epilepsy, but do not give it the prominence of the preceding authors. They regard the Ammon's horn sclerosis as a developmental defect, and a local expression of a general condition that accompanies other brain lesions. Binswanger suggests that epilepsy can have its origin in (a) demonstrable anatomical changes; or (b) in fine molecular disturbances of the nerve-cell which cannot be demonstrated; or (c) in irregular evanescent disturbances of the metabolism of the nerve-cell. Oppenheim regards the glial changes in the outer cortical layer and in the cornu ammonis as a sequence rather than a cause of the epilepsy. This view is also shared by Robertson. In the brains of seventy epileptics reported by Bratz, twenty-five presented sclerosis of the cornu ammonis. The minute changes consisted in atrophy of the pyramidal cells of the third layer and the stratum granulosum. Many presented pigment deposits. The atrophied and destroyed structures appeared to be replaced by a dense network of neuroglia fibers. The author was not able to make distinction, as regards this matter, between the cases of long duration and those of more recent development. He does not believe these changes are secondary, but is uncertain about their place in the etiology of epilepsy. Tedeschi found diffuse gliosis in a case of infantile cerebral palsy complicated by epilepsy, the gliosis being on the affected side only.

The author leans to the view that epilepsy has its origin in a gliosis. Eurich describes pronounced neuroglia proliferation in the outer cortical layer without other tissue changes. Lorenz unfortunately failed to find any minute lesions of the brain cortex in his study of status. Weber, on the contrary, observed numerous minute extravasations of blood scattered throughout the brain and medulla with partial destruction of nervous tissue, to which he ascribes the direct causes of death. In a recent study of status, Muchand fails to note any neuroglia changes, but finds some alterations in the cells of the cortex, consisting mainly in chromatolysis and infiltration of the cortex with leucocytes.[1]

It will be seen from the foregoing that the writers on the pathology of epilepsy in general, during the past twelve years, regarded it as a disease of the cortex. The weight of opinion leans strongly to the nerve-cells as presenting the lesion of greatest importance. It is to be noted, however, that the origin of epilepsy in an overgrowth of the neuroglia still has its advocates. This idea, which in recent years has been distinctly on the wane, has received an added shock in the work of Starlinger, Prus, Hering, Bischoff, and others.

[1] Wright, in some observations on a fatal case of potassium bromide poisoning (Brain, 1898, page 186), feels justified in concluding that potassium bromide is capable of destroying the proper tissue of the gray matter of the cerebral cortex, if taken in sufficient quantity; and that the bromides act upon, and degeneration begins in, the protoplasmic processes of the cell rather than in the cell body.

Notwithstanding the fact that the author endeavored to control his observations, we cannot, for several reasons, accept them as final. We observe that the temperature of the patient for more than two days immediately prior to death was very high, reaching at one time 108.4° F. Further than this, the autopsy was not done until twelve hours after death, the brain being immersed entire in formalin, two factors which rendered the tissue unfit for staining after the Nissl method, especially where judgment of the minute cell-changes was pending. The Nissl method following formalin fixation is inconstant. The use of the silver stains cannot be regarded as in any sense ideal in the investigation of neuro-pathological changes, but when the method is used on material, twelve hours post mortem, which has been subjected to a temperature of 108.4° F., plus the toxic products of a pneumonia, we can hardly regard the results and conclusions seriously.

Prus has recently undertaken the demonstration of the paths over which the epileptic fit generalizes, with special reference to the involvement of sensory or motor paths in the conduction of the epileptic impulse. He dissents from Unverricht's so-called law of irradiation in epilepsy. Unverricht, after employing electrical irritation of the cortex and obtaining a consequent fit, suggests that the cortex is the point from which the fit begins and spreads, and that the succession of the spasm, as regards its spread through the motor cortex is exactly comparable to the succession of waves that follow the casting of a stone into a quiet pool of water. Prus, after one hundred experiments on dogs, declares that the generalization of the epileptic fit, as seen after electrical irritation of the cortex, follows no general law. Unverricht's theory has also been criticised by Binswanger and others.

In order to determine the paths over which the epileptic impulse travels from the cortex to the periphery, Prus severed the pyramidal tract at various levels on one and both sides from the internal capsule to the spinal cord. Notwithstanding this, however, the development and generalization of the fit were not interfered with on electrically irritating the cortex. The author concludes that there must be some other than this well-known motor path between the cortex and the periphery over which the epileptic impulse travels. His next experiments were undertaken to prove the location of these tracts; the assumption being that, if their position can be known, the path of the epileptic seizure is determined. For this purpose he cut into the peduncles on both sides, severing all except pyramidal fibers, and stimulated the cortex. No fit resulted. If the motor centers were stimulated the corresponding muscle-group contracted, but no epileptiform manifestations followed. The conclusion is reached that neither the pyramidal tracts, the fronto-bridge tract nor the temporo-occipito-bridge tract take any part in the transmission of the impulse in epilepsy; that the impulse is transmitted by means of tracts which lie entirely without these, and which Prus calls extra-pyramidal tracts; and that these tracts run in the dorsal portion of the peduncles.

In order to elucidate the question of the essentially sensory or motor character of the spasm in epilepsy, the author painted

the cortex with various agents which impair the sensibility of the sensory elements. The bromides produced diminished irritability of the cortex. When cocaine was applied locally no fit could be produced by attempts at irritation of the motor cortex, but the corresponding muscle-groups would still respond. He holds, therefore, that the starting point of the fit in cortical epilepsy is either in the end tufts of the sensory nerves in the cortex, or in the sensory cells of the cortex, and not in the motor cells or fibers. His ultimate conclusions were as follows: (1) that the transmission of the impulse in epilepsy is through the extra-pyramidical tracts which transmit motor reflex impulses; (2) that the sensory elements of the cortex must be irritated in order to produce the fit; (3) that the fit appears to be a complicated reflex phenomenon.

Hering, more recently, has substantiated some of the more important points in the work of Prus. He has also demonstrated the existence of the extra-pyramidal tracts and produced epileptiform fits after severing the motor tracts on both sides. Probst has also added important corroborative evidence in affirming the existence, by experiment, of other than the pyramidal tracts which are capable of transmitting motor impulses.[2] The effect of the bromides and cocaine, when applied locally, in impairing the sensibility of the sensory elements of the cortex, has more recently been demonstrated by Rossi. Quite recently Ossipow has demonstrated the occurrence of violent contractions of some of the abdominal viscera during the epileptic seizure; a fact of some interest, since it reflects the essentially reflex character of the spasm.

In reviewing thus briefly some of the more important work of the past 14 years, we find that our knowledge of the exact nature of epilepsy is now established on a broader and firmer foundation, notwithstanding the fact that a multitude of questions still remain unsolved and still press for solution. The

[2] The further physiological and anatomical demonstration of these tracts has been accomplished by Redlich (Monat. f. Psych. u. Neurol. Bd. V, 1899), Rothman (Zeitschr. f. klin. Med., Bd. 44), and V. Bechterew (Neurolog. Centralbl. No. 23, 1897).

The fasciculus sulco-marginalis descendens and the fasciculus rubrospinalis transmit certain motor impulses which partake of a reflex character.

cortical origin of most epilepsies has been firmly established by pathological observation and psychological experimentation. The neuroglia hyperplasia in epilepsy is now almost constantly found. With improved methods and technique it will probably be demonstrated in every case of any considerable duration. At the same time the minute changes in the nerve-cells of the cortex are coming to the fore and demanding recognition. The increasing importance of these changes tend to the relegation of the neuroglia hyperplasia to a secondary place.

Gross Anatomical Lesions of Status.

Since status is but the climax of epilepsy proper, we shall, of course, find in status cases all the gross anatomical changes in skull, brain and membranes incident to epilepsy in general. These will vary from slight stigmata of degeneration (so called) to very positive deformities of the cranium and its contents. In the present state of our knowledge, these changes are to be regarded as secondary. In those epilepsies following in the wake of infantile cerebral palsy, for example, the gross lesions can only be considered as conditions rendering the patient especially susceptible to the influence of the various toxic and autotoxic agents, which are undoubtedly the prime inciters of the pathogenesis of all the epilepsies. If the skull is injured by trauma it encroaches upon a growing brain; if a hemorrhage has occurred in some portion of the brain substance a positive destructive lesion is the result. Meningeal hemorrhages generally result in meningeal adhesions forming in the later life of the individual. All these create irritable foci or areas of the brain especially susceptible to irritating influences.

Pathological states, such as those just enumerated, result also in permanent lesions, which are evident in the adult in the form of the irregularly shaped skull with abnormally thick external and internal plates. A skull of this sort may present many other abnormalities, such as unusual thickening in whole or in part (exostosis), or extreme density throughout, with more or less complete obliteration of the diplöe and an abnormal distribution of the meningeal furrows. Considerable thickenings of the meninges may present themselves with abnormal adhesions to skull and brain surface, while the brain itself may show areas

of localized sclerosis and atrophy, or perhaps an atrophied area, the direct result of an old hemorrhage or embolus, or the lesion of an infantile cerebral palsy. Gross lesions of this sort are not infrequently found post mortem in epileptics.

The developmental defects of the epileptic, some of which are comprised above, are of undoubted import from an etio-pathological standpoint, since they tend to the production of an unstable, nervous organism. This point is of great importance, since it concerns prognosis. The result of treatment will depend to a large extent upon how far the organism has been diverted from normal channels in its development. In this connection we believe Ohlmacher's interesting observations on the lymphatic diathesis and thymic hyperplasia in epilepsy, are to be looked upon as expressions of mal-development (stigmata of degeneration), organic anomalies which may seriously impair the power of the organism to resist irritants and toxic elements. Roncoroni's work on the anomalous development of the cerebral cortex of epileptics is to be looked upon in the same way.

The character of the more recent gross lesions found after a period of status depends largely upon the particular stage in which the patient dies. While the changes in the early stages are congestive and hemorrhagic in character, in the later period, lesions referable to exhaustion and anæmia with some evidences of a mild inflammatory action are superimposed. These are manifest in the marked congestion of the pia-arachnoid and its slight adhesion to the brain surface, with occasional areas of slight hemorrhage between the meningeal layers; and a greater or less diminution in the consistence of the brain substance itself with anæmia or congestion, as the case may be. Gross lesions of this type are also to be looked upon as a result rather than a cause of status.

While it is possible that an epileptic may die of meningeal apoplexy—and authentic cases are occasionally recorded—it is nevertheless a fact that it is extremely rare for an epileptic to die from any form of apoplexy, and rarest of all apoplexy from meningeal hemorrhage. Gross anatomical lesions of this character are therefore comparatively rare following status.

Although a few status cases occur in the plethoric and obese, yet the greater majority suffer from general malnutrition, and

the general appearance of the body after death is usually that of more or less marked emaciation. Bruises and scars from injury in convulsions are common. More or less extensive bed-sores are found on the nates, heels, scapulæ and elbows. In fatal cases the sites of bromide injections may appear either as large rose-colored spots or areas of actual sloughing. The skin usually presents multiple petechiæ (hemorrhage) on the face, occurring most commonly about the eyes and at the angles of the jaws; less frequently on the neck and chest, and occasionally on the extremities, depending upon the severity of the status. Post-mortem discoloration occurs rapidly, especially if the patient is plethoric and dies from acute exhaustion. In such cases the body may be almost completely discolored in consequence of decomposition within a few hours after death. Rigor mortis is always delayed in cases dying from "acute status," and rapid in cases with prolonged stuporous periods. In typical cases, however, it is undelayed. In acute status the body temperature often does not reach normal until from 3 to 12 hours after death.

In early fatal status the dura is often tense from fluid distention. In prolonged fatal status, in which a comatose stage is present, the dura distention is absent. In the acute stage there is marked œdema of the pia-arachnoid, due to the greatly increased arterial pressure during the seizures and the obstruction to the return venous flow. The pia is frequently adherent, slightly thickened and congested. There is rarely any pial opacity or marked evidences of tissue formation as a consequence of the status. In a few prolonged status cases, dying some weeks after the acute onset of the condition, the pia presents a low-grade inflammation. The brain substance is congested or anæmic, depending upon the stage in which death occurs—congested in the early stage, anæmic in the later.

The abdominal viscera show more or less severe congestion, depending also, though to less extent than the brain tissues, upon the stage in which death occurs. The lungs are generally œdematous if death occurs during the convulsive stage; if in the stuporous stage, there is hypostatic congestion, or areas of consolidation; if tuberculosis antedated the status, an extension of this process may be found. The heart is usually flabby and in diastole; the walls are soft, friable and frequently show acute

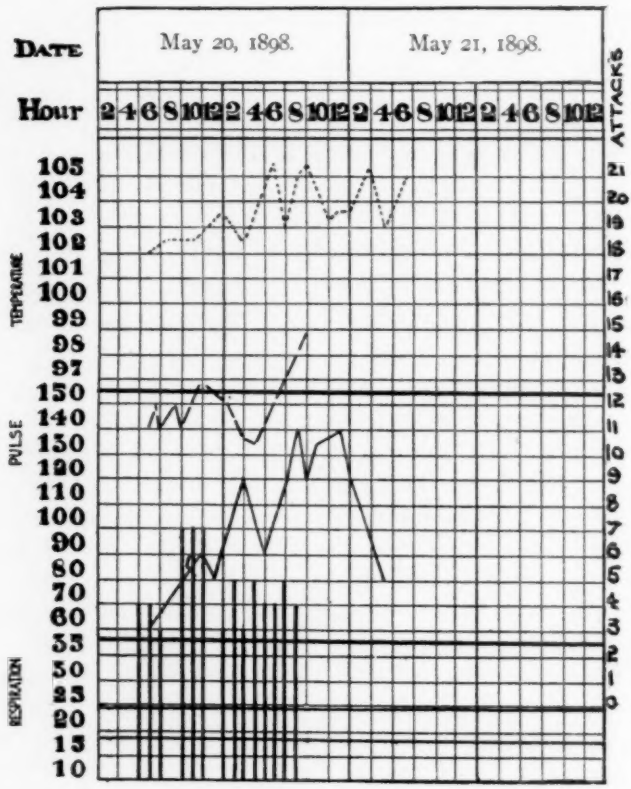
degenerative changes. The heart muscle has been known to rupture during status. The myocardium is usually congested and evidences of active inflammation are not infrequently found. The liver is always congested, and, on section, the exuding blood is dark and tarry in consistence. More hemorrhages are found in the liver substance than in any other viscera except the spleen. Fracture of the liver, as a consequence of the violent spasms of status, has been observed. The spleen is always enlarged in severe status cases, sometimes twice the normal size, and often studded with infarcts. A section of the organ shows the tissue friable and diffluent. The kidneys are usually pale on section; numerous small infarcts may stud the surface in rare cases. The cortical striations are often poorly defined.

Authors differ very widely as to the conditions and lesions presenting in the ductless glands. As for ourselves, we find the morbid changes in the epilepsies no more abnormal than in autopsies of other neuropathic states where bodily and mental degeneracy is equally prominent in evidence.

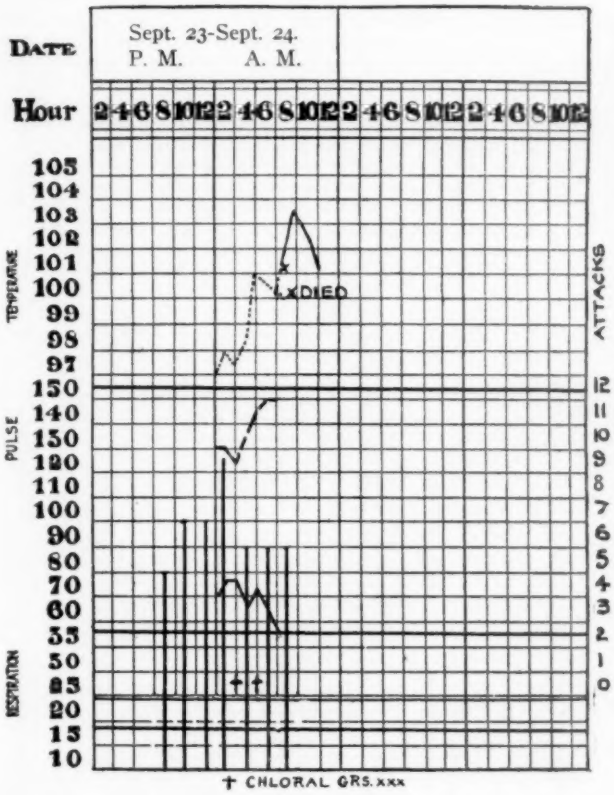
(*To be continued.*)

EXPLANATION OF THE CLINICAL CHARTS:—The general scheme is to show the alterations and relationship subsisting between the fever, pulse and respiratory curves in a few illustrative cases of status epilepticus. The changes in the three curves were noted in periods of two hours. The curves take their reading standard from the left as in ordinary fever charts.

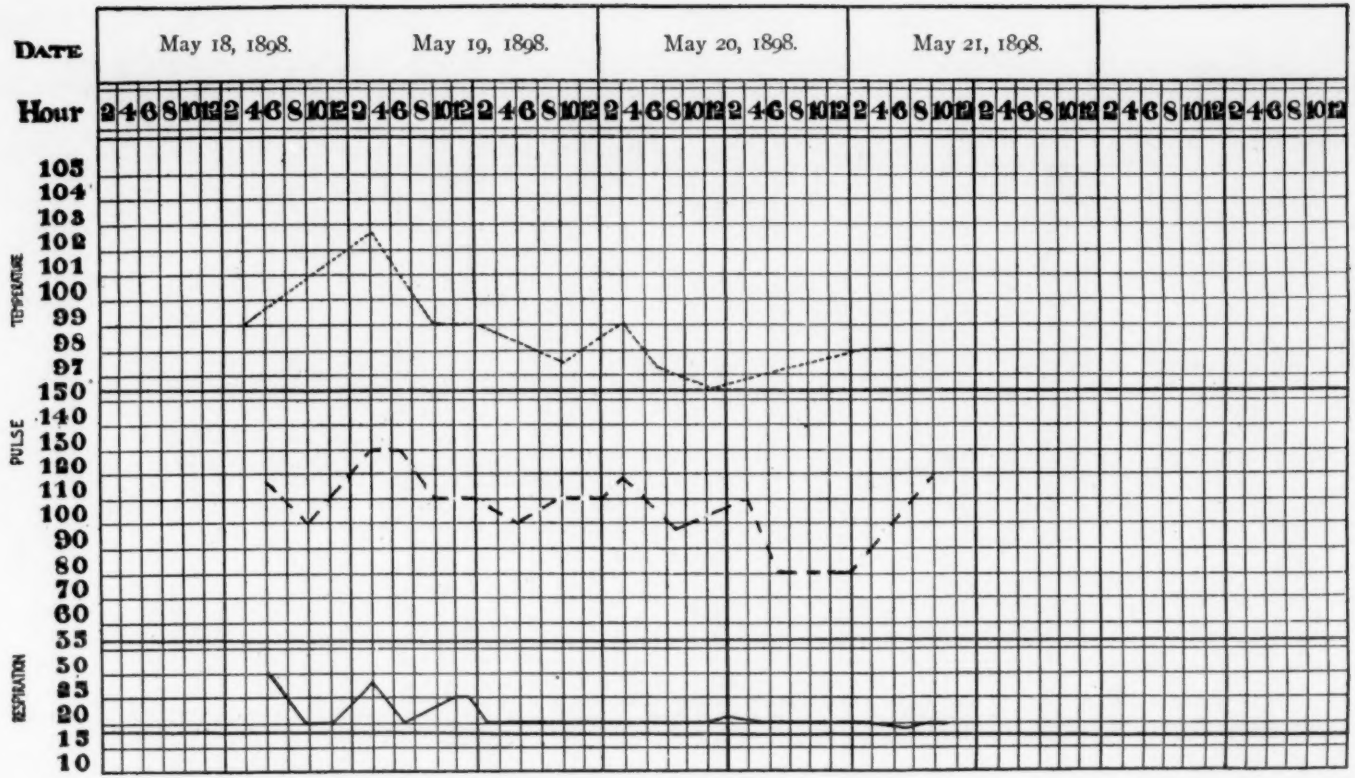
The hourly record of epileptic attacks is represented by solid columns, their reading standard is at the right of the chart. The columns are prolonged to the base line of the chart in order to aid the eye in following the record to advantage.



CLINICAL CHART, CASE I.—Mary H., aged 23; G. M. idiopathic epilepsy from 5 years of age. Typical, acute fatal status with an abbreviated stuporous stage. Note the step-like increase in the cardinal curves in spite of sedative treatment, also the cause and effect relationship between the seizures and the cardinal curves.

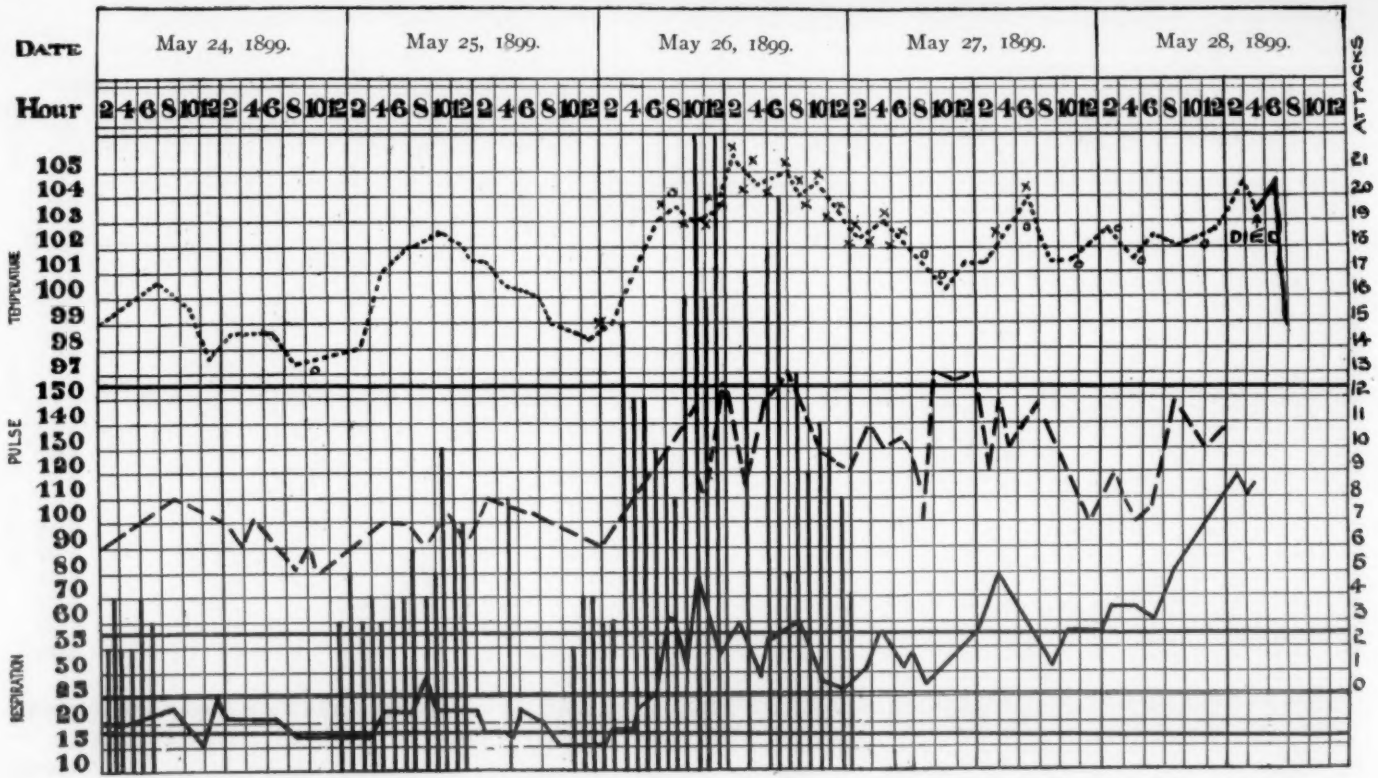


CLINICAL CHART, CASE II.—Charles M., aged 16; idiopathic G. M. from 2 years of age; third status period. The acute status here recorded was provoked largely by an accidental reduction of sedatives. Death in the convulsive stage. The post-mortem rise in temperature is well marked.

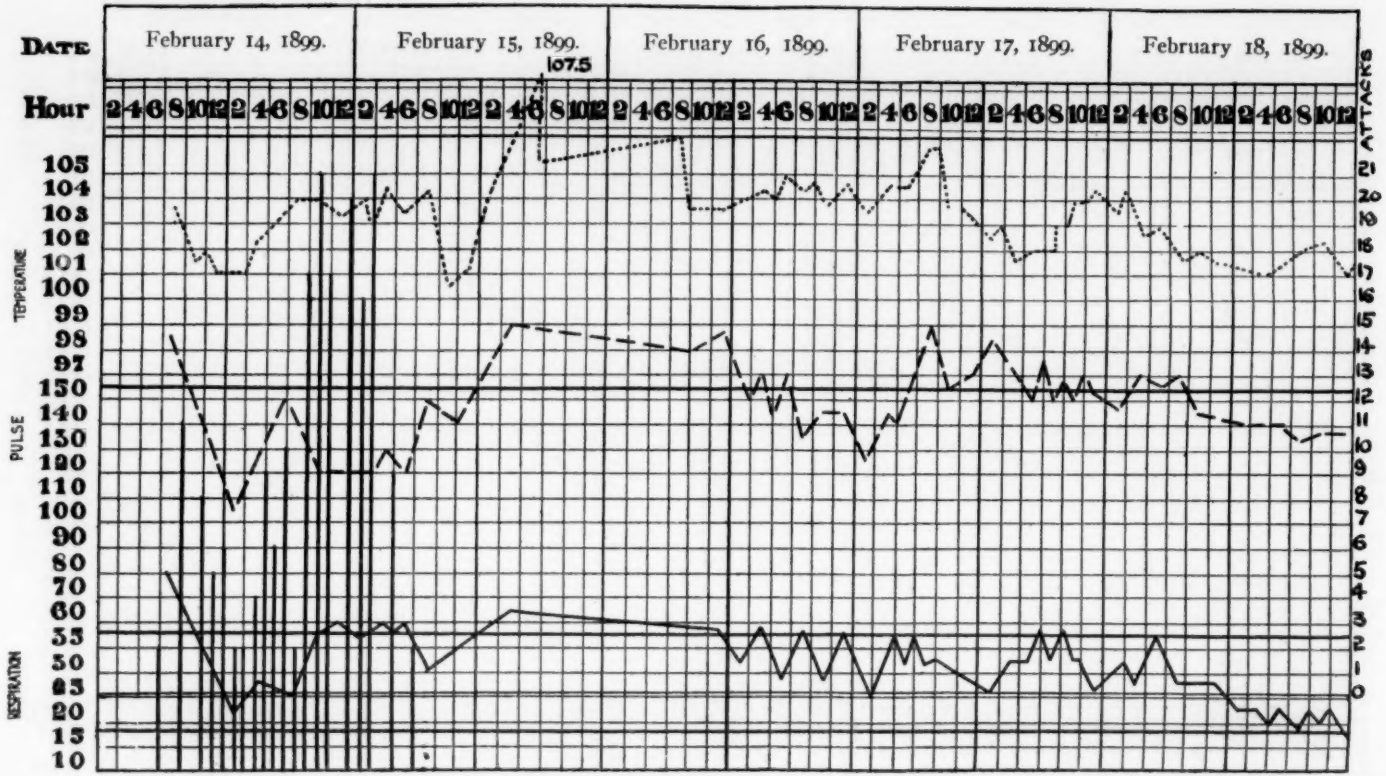


CLINICAL CHART, CASE III.—Emma C., aged 10. Psychic idiopathic epilepsy began at 3½ years of age, caused by erysipelas. Patient had about 40,000 psychic ("absences, vertigoes or fainting spells"), attacks in 6½ years. Attacks consist of an inclination of the head to the left; the patient stands still, the eyes are closed for a few seconds, the condition is then terminated by a long-drawn sigh; the patient immediately after resumes her work or play as before. Frequently 120 attacks occur daily without ill effects. The chart shows a mental equivalent of status in which no psychic attacks occurred, the first free period in two years; the patient remained in a dazed mental state throughout. The state was terminated by the gradual reappearance of the usual psychic attacks.

the left; the patient stands still, the eyes are closed for a few seconds, the condition is then terminated by a long-drawn sigh; the patient immediately after resumes her work or play as before. Frequently 120 attacks occur daily without ill effects. The chart shows a mental equivalent of status in which no psychic attacks occurred, the first free period in two years; the patient remained in a dazed mental state throughout. The state was terminated by the gradual reappearance of the usual psychic attacks.

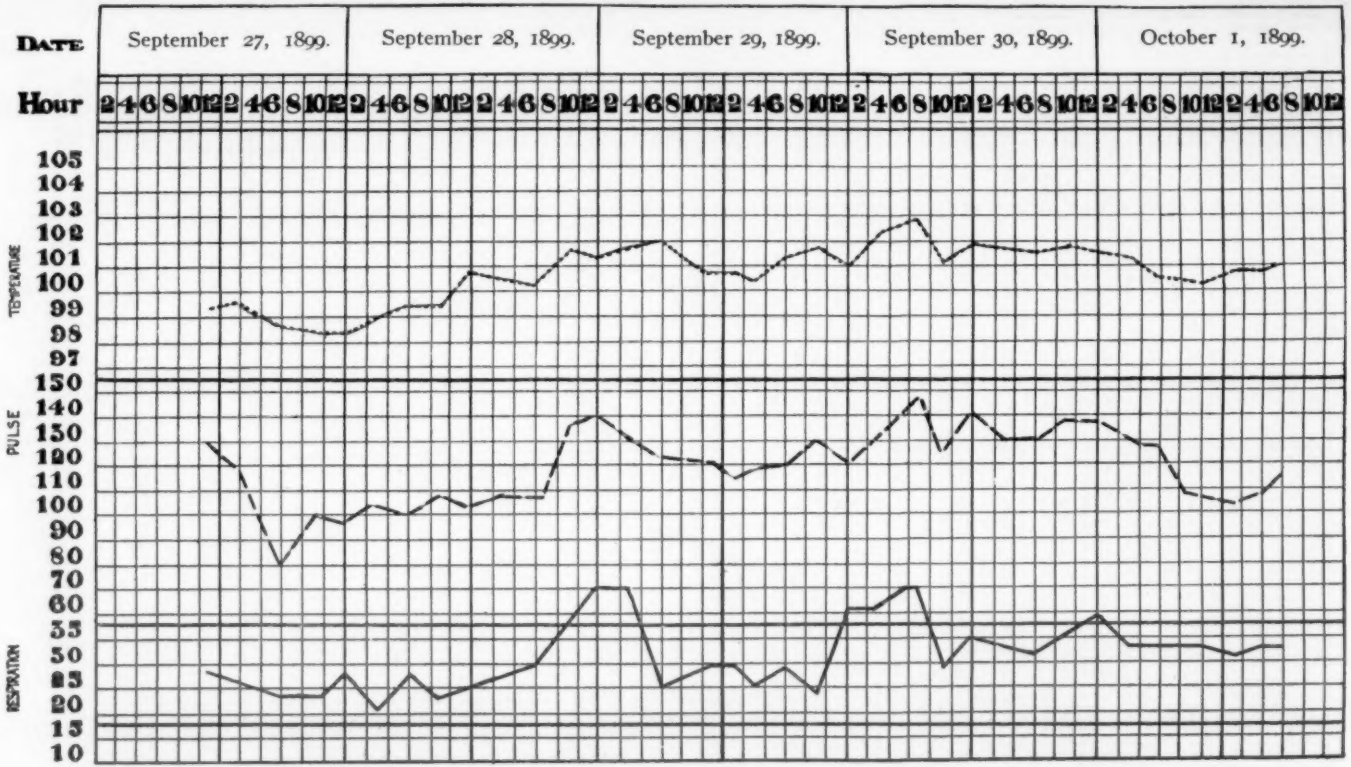


CLINICAL RECORD, CASE IV.—Fannie D., typical fatal status =: x = baths, o = alcoholic stimulants. Note post-mortem rise of temperature in the stuporous stage.

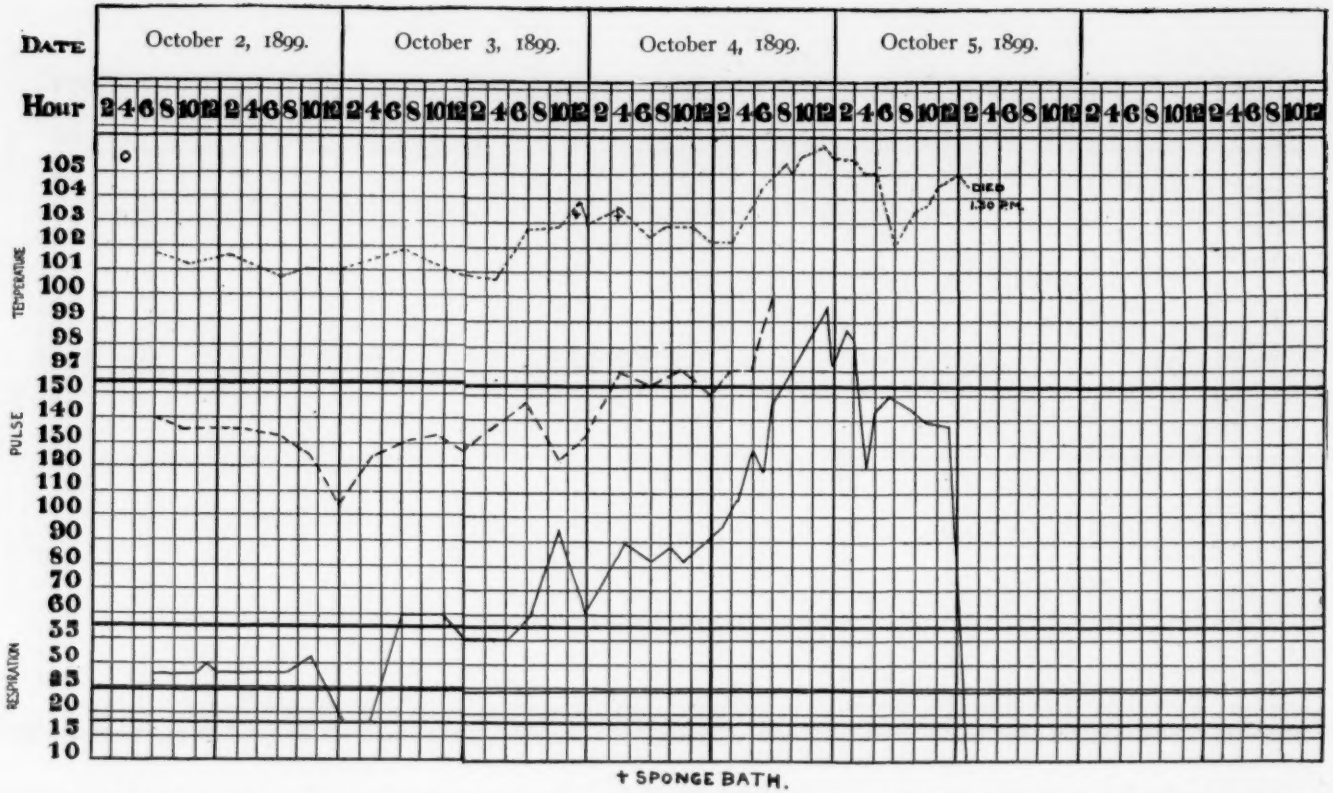


CLINICAL RECORD, CASE V.—Fannie D., aged 13; idiopathic G. M. from 3 years of age. Forty attacks occurred without rise in temperature on February 13. This typical status period comprised 228 G. M. attacks in 55 hours and ended in recovery. Chart discontinued from 6 P. M. February 15 to 8 A. M. February 16. See photographs of same case.

6 P. M. February 15 to 8 A. M. February 16. See photographs of same case.

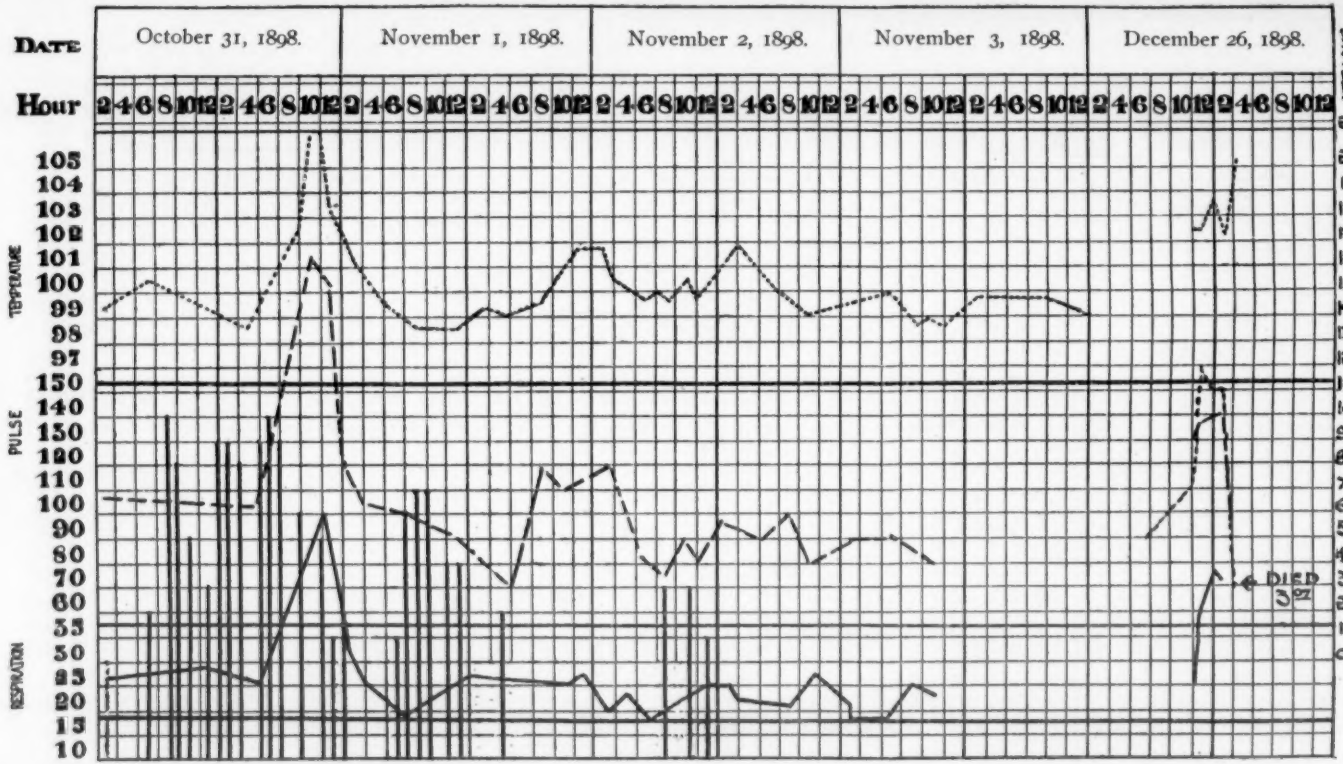


CLINICAL CHART, CASE VI.—Mary S., aged 21 years; hemiplegic epileptic from 8 months old; attacks usually G. M. with an order of muscular invasion interspersed by psychic attacks of sudden fury and homicidal assault. Usually the latter attacks last 20 minutes followed by deep sleep for an hour or more. No convulsive phenomena or febrile disturbance occur with the isolated convulsive or psychic attacks.

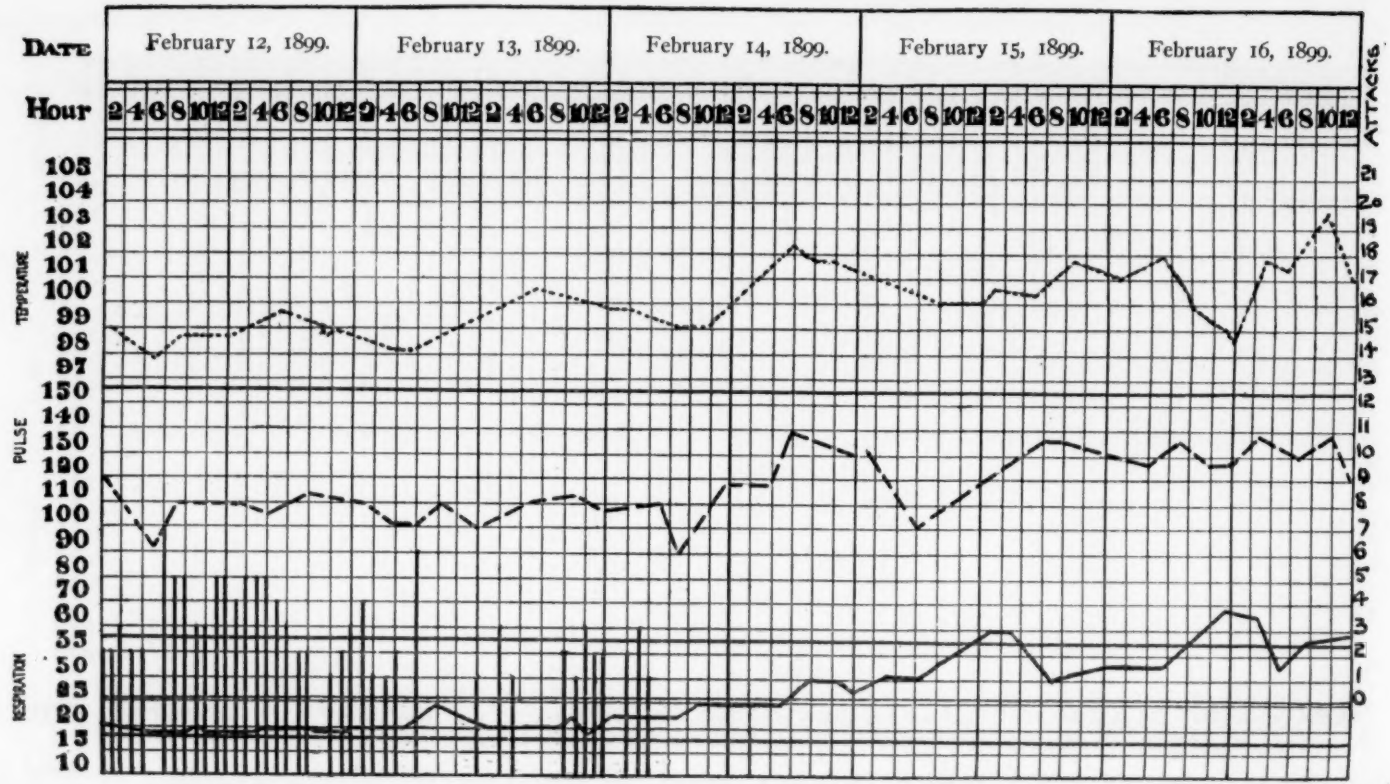


For 7 days (September 27–October 3), patient was in a state of intermittent attacks of fury (epileptic), each exacerbation of violence being similar in all respects to the isolated psychic attacks previously noted. Typical status coma and stupor then developed and ended fatally October 5. Autopsy was negative aside from the typical microscopical findings noted in the pathology. The case is one of psychic status equivalent.

similar in all respects to the isolated psychic attacks previously noted. Typical status coma and stupor then developed and ended fatally October 5. Autopsy was negative aside from the typical microscopical findings noted in the pathology. The case is one of psychic status equivalent.

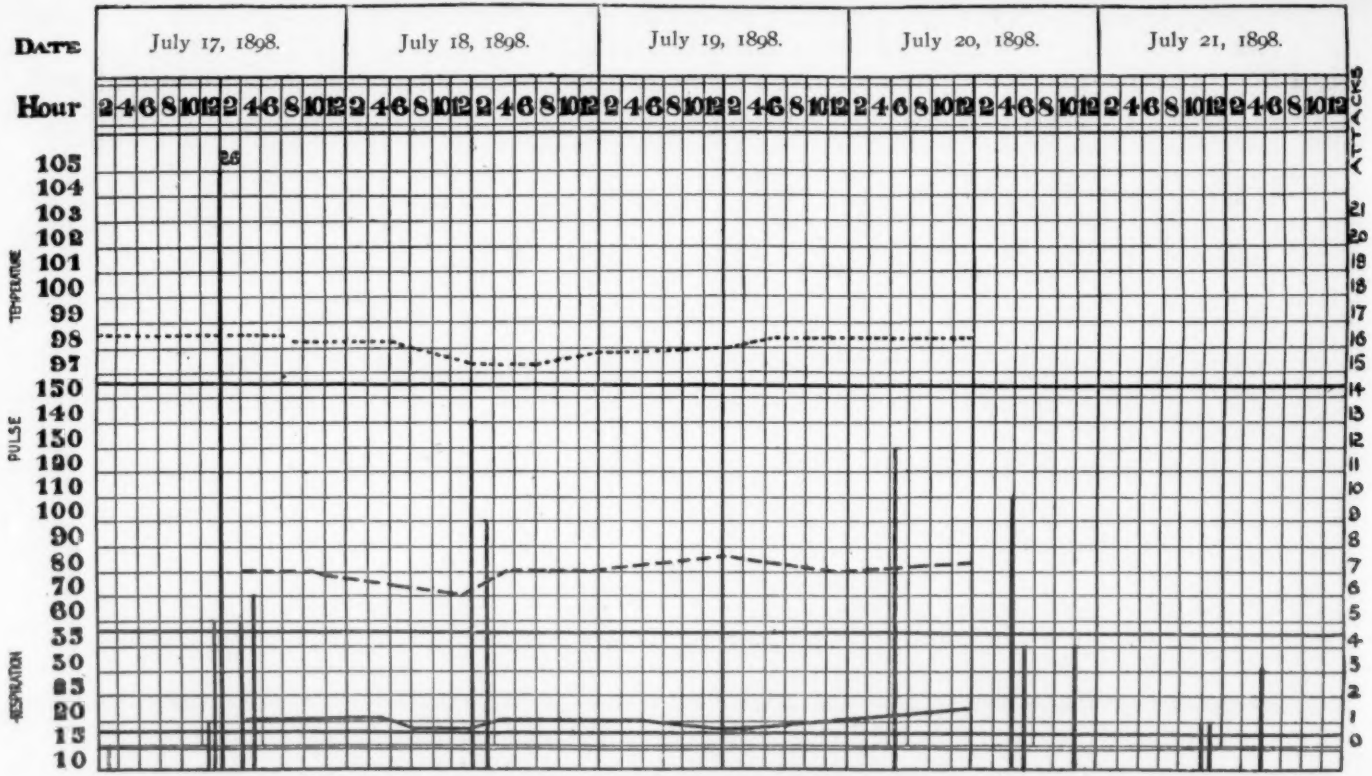


CLINICAL CHART, CASE VII.—William R., aged 20; G. M. epilepsy from age of 16 years. The seizures forming the first status (Oct. 3, 1898), were statuesque or tetanoid in character, only tonic spasm being present. The second and fatal status, occurring the following Dec. 26, was an acute attack; the convulsions were G. M. in type. Death resulted from respiratory failure; heart continued action for 1½ minutes after respiration ceased. The seizures in the last and fatal period are indicated by a dotted line.



CLINICAL CHART, CASE VIII.—Fred. D., aged 23 years; idiopathic epilepsy. Beginning status controlled by sedatives.

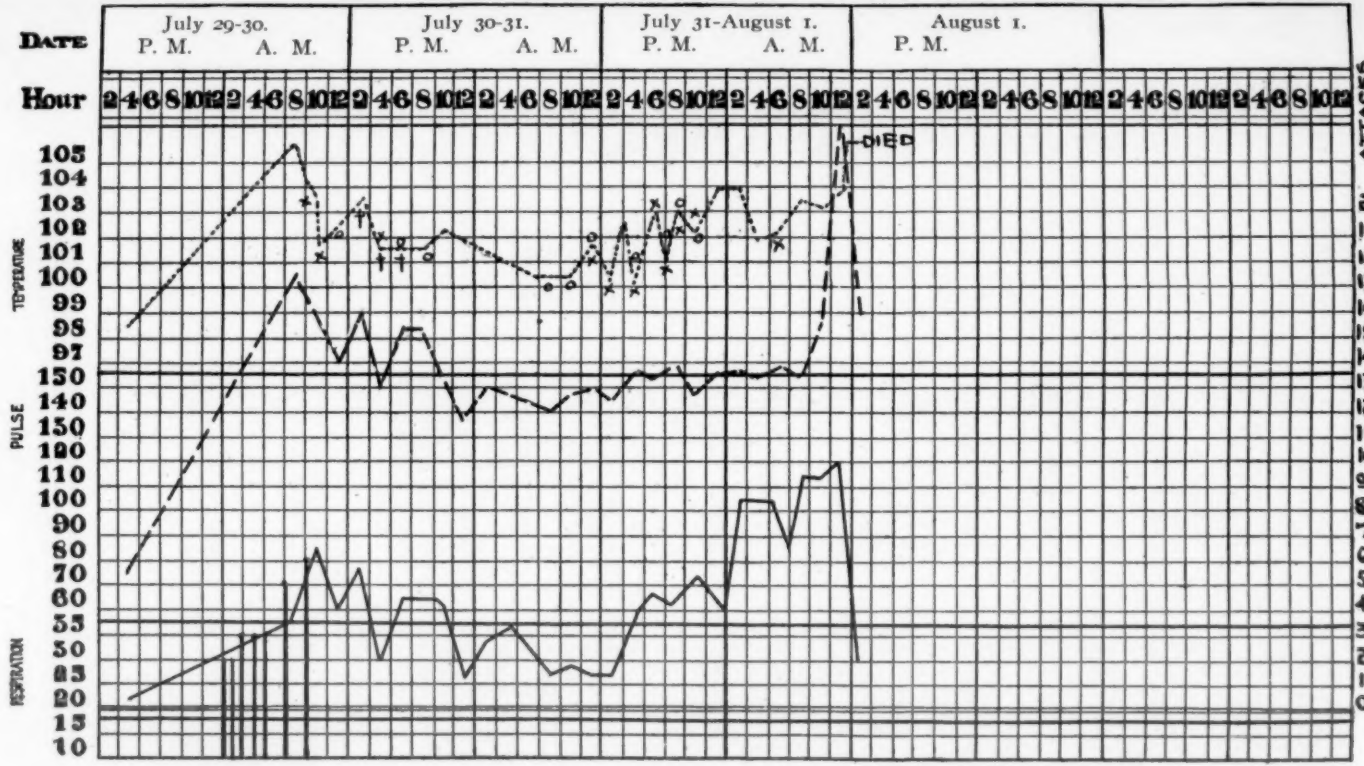
CLINICAL CHART, CASE VIII.—Fred. D., aged 23 years; idiopathic epilepsy. Beginning status controlled by sedatives.



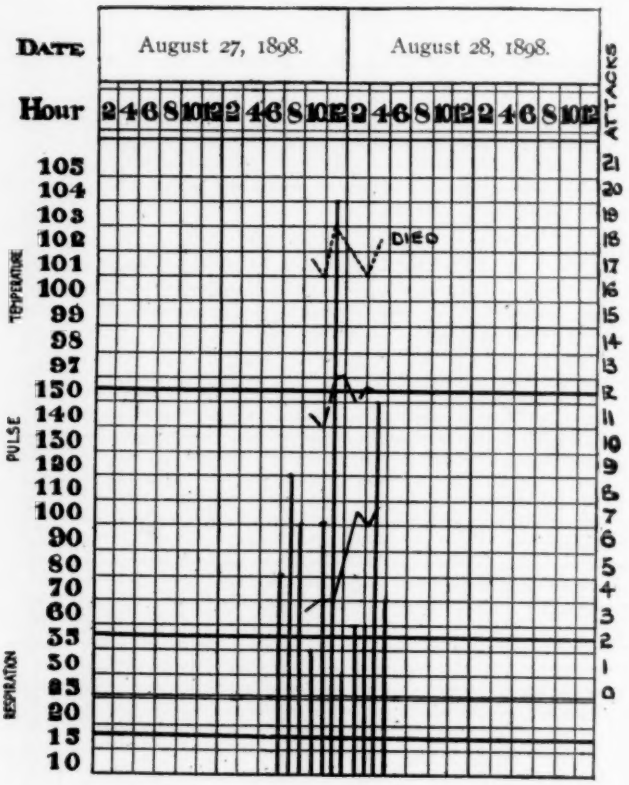
CLINICAL CHART, CASE IX.—Ida N., aged 17 years, major hysteria for 3 years. The clinical record shows the very marked difference between the cardinal curves in status hystericus and status epilepticus. The accompanying annual register of attacks shows 4 marked periods of status hystericus. The periods are noted by (x) in the annual register of hysterical convulsions. The case ultimately fully recovered.

IDA N.—GRAND HYSTERIA.

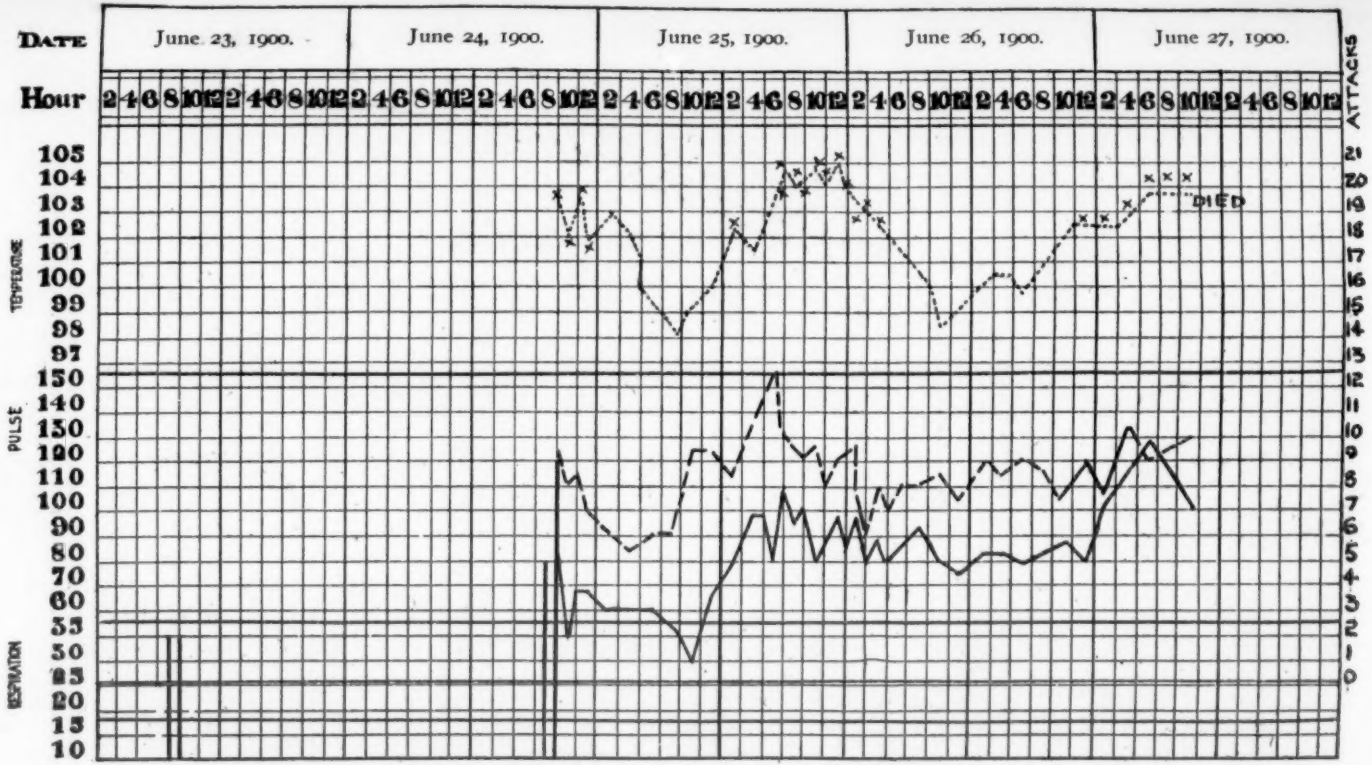
898	1	2	3	4	5	6	7	8	9	10	11	12	13	14	5	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	Total.
J...	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
F...	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
M..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
A...	..	..	..	..	..	..	..	..	..	..	..	..	.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
M...	..	..	..	.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
J....	..	..	..	..	..	..	..	..	..	..	..	..	..	Ad. P 1	G 1	..	..	..	..	..	..	..	..	..	..	..	G 1	..	..	..	..	3
J...	..	..	..	..	Ps 4	G 2	..	..	Ps P 9.1	Ps P 11.3	Ps 15	..	Ps 1	..	..	⊙	Ps 38	Ps 22	Ps 1	Ps 30	Ps 6	Ps 13	Ps 4	Ps 6	Ps 1	Ps 4	Ps 14	Ps 2	Ps 3	Ps 3	Ps 1	194
A...	..	..	Ps 8	Ps 30	..	Ps 5	Ps 2	..	Ps 7	..	..	Ps 6	..	Ps 2	P 1	..	G 1	..	P 1	P 2	..	P 4	..	..	..	..	..	⊙	P 4	P 95	P. G 32.5	205
S...	P. Ps 24.1	P G 7.1	P 2	P 1	P 2	..	..	⊙	G 202	G 40	G 29	..	..	..	..	..	G 2	G. P 10.2	..	..	..	P. G 1.1	..	..	..	..	..	..	P 1	..	..	326
O...	..	Ps 4	..	..	..	P 26	P 1	..	..	⊙	G 5	P 14	G 44	..	..	..	P 5	..	G 1	..	P 1	G 1	..	P 4	G 5	..	..	..	..	..	..	111
N..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
D..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
																																839



CLINICAL CHART, CASE X.—Josephine D., aged 20; epileptic idiocy; epileptic from childhood. Convulsive stage is here shown only in part. 62 attacks occurred in 4 hours prior to beginning rise in pulse, temperature and respiration at 4 P. M., July 29, as shown in chart. The preliminary fall before the fatal rise in the curves with stuporous periods was probably due to the baths and restoratives: the two maximal points in cardinal curves in status charts may often be accounted for in this way.



CLINICAL CHART, CASE XI.—Walter S., colored, aged 24 years; idiopathic G. M. epileptic from 6 years of age. Status precipitated by accidental lowering of sedation.

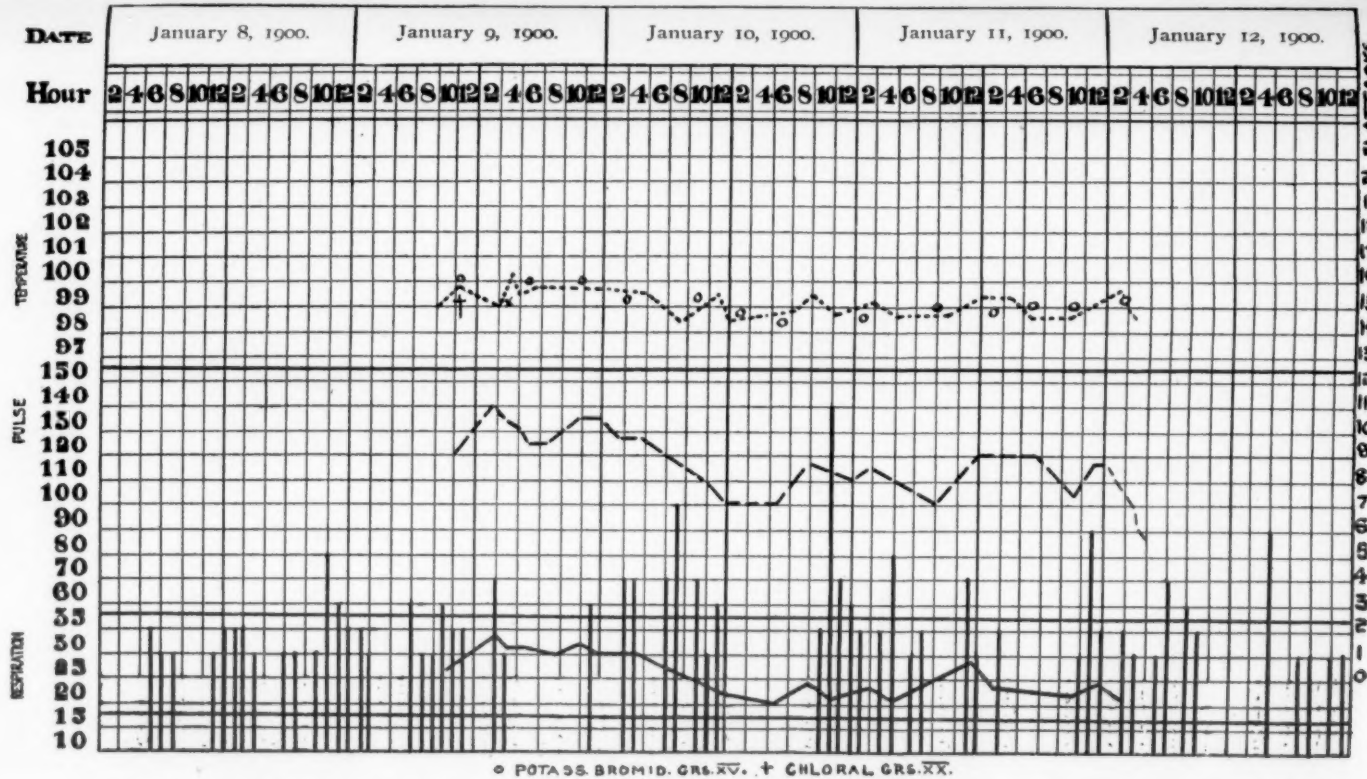


CLINICAL CHART, CASE XII.—Clarence B., aged 30 years; G. M. epilepsy. Note the disproportion between the convulsive and stuporous stage. 14 G. M. attacks precipitated the fatal ending. The baths account for the atypical curves in the stuporous stage. Autopsy was negative except the microscopical findings noted in the pathology.

The annual register of epileptic seizures in this case shows the marked tendency for the formation of serial periods, the harbingers of status epilepticus.

CLARENCE B.—ANNUAL REGISTER OF EPILEPTIC ATTACKS.

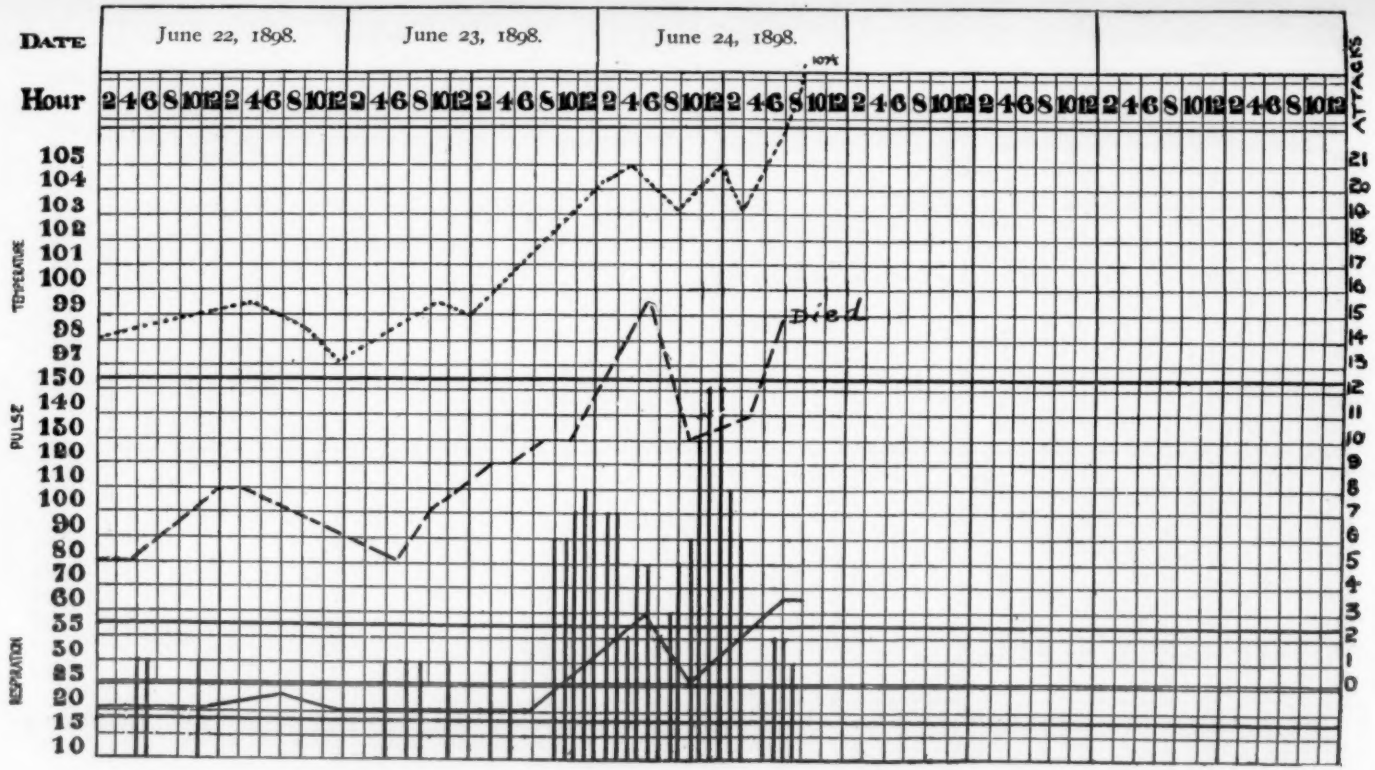
1900	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	Total
J....	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
F....	..	..	..	..	..	.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	Ad.	..	..	..	
M....	..	..	..	..	..	..	..	..	G 1	..	..	P 1	P 1	..	..	P 1	G 4	G 6	G 5	G 5	G. 8.7	P G 2	..	..	..	..	..	..	..	..	..	41
A....	..	..	..	..	..	..	..	P 2	P 3	..	P 3	P 1	P 3	P 2	P 1	P 3	P 4	P 1	..	P 1	P 8	..	..	P 1	P 1	..	..	..	..	..	..	29
M....	..	..	..	..	..	P 1	..	..	..	..	P 1	..	..	P 1	..	..	..	..	P 1	..	..	..	..	..	..	..	P 1	..	..	P 1	..	P 4	10
J....	P 2	..	..	..	..	..	..	..	..	..	..	P 6	G 5	G 3	G 6	..	..	..	..	..	..	..	..	G 14	..	..	Died	..	..	..	..	..	36
J....	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
A....	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
S....	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
O....	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
N....	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
D....	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
																																116



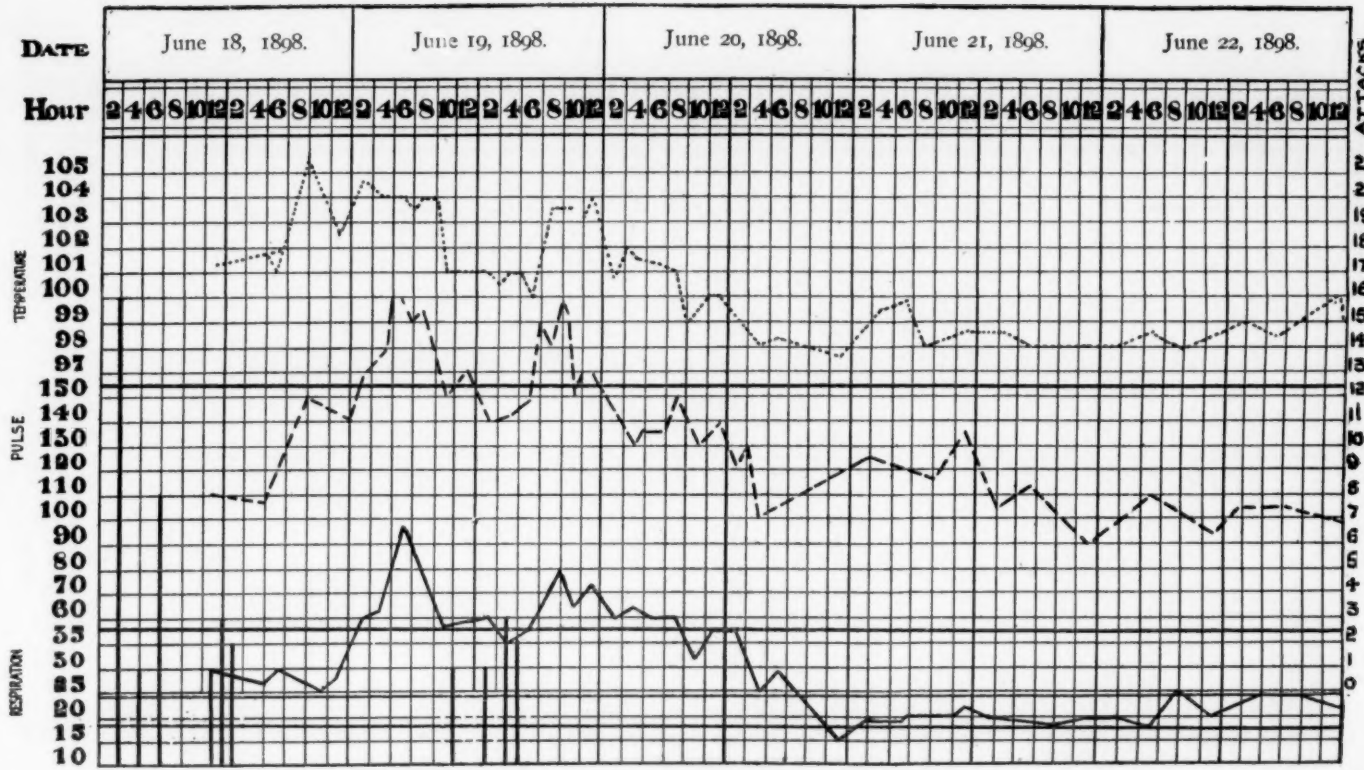
CLINICAL CHART, CASE XIII.—Isabella D., aged 8 years, idiopathic epileptic from 3 years of age. An illustrative serial period which is often a herald of status. Prompt treatment in this period is most effective in aborting status. The tendency to formation of mild status and serial periods is well shown in the annual register of seizures accompanying this clinical chart.

ISABELLA D.—ANNUAL REGISTER OF EPILEPTIC SEIZURES.

1900	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	Total.
J...	..	..	..	..	..	Ad.	..	G 16	G 44	G 47	G 23	G 20	G. P 4.6	P 2	P 5	P 2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	P 2	169
F...	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	G 1	..	..	..	..	..	..	..	..	..	1
M.	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	Ps 1	..	..	..	..	..	..	..	..	..	..	1
A..	..	..	..	..	..	..	..	..	G 1	..	P 3	Ps 6	Ps 5	Ps 18	Ps 11. G 2	G 20	G 16	G 12	G 15	G 14	G 8	Ps 3	Ps 3	G 1	..	..	..	..	..	..	..	138
M..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	G 1	..	..	..	..	..	..	..	1
J...	..	..	..	Ps 1	..	..	..	..	..	..	..	..	..	..	..	..	G 1	..	..	..	G 1	..	..	..	..	G 1	G 1	..	..	..	..	5
J...	Ps 9	Ps 6	Ps 7	G 7	G. P 6.4	G 6	P 9	Ps 12	Ps 12	Ps 6	Ps 5	Ps 4	Ps 6	Ps 7	Ps 5	G 3	P 3	G 1	P 3	G 6	G 1	..	..	..	P 2	..	..	..	..	P 2	..	132
A..	..	..	G 2	G 2	..	..	..	..	..	..	Ps 2	..	..	..	..	..	Ps 1	..	..	..	..	..	..	..	..	..	..	..	..	..	P 2	9
S...	..	..	..	..	..	..	..	..	..	..	G 1	G 2	G 1	G 2	G 3	G 4	G 5	G 4	G 7	G 12	G 16	G 21	G 24	G 26	G 22	G 11	P 1	P 1	..	..	..	163
O...	P 1	..	..	..	..	..	P 1	..	G 1	G 2	..	G 1	G 1	Ps 1	..	.	..	..	..	..	..	..	..	..	G 2	G 1	..	..	..	..	..	11
N..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	.	..	..	..	..	..	..	Ps 1	..	G 1	..	2
D..	P 2	G 4	G 4	P 7	P 6	P 6	P 5	Ps 6	Ps 9	P 9	P 24	P 11	P 12	P 18	P 19	P 18	P 17	P 21	P 16	P 18	G 22	P 7	..	..	P 4	P 3	P 2	P 11	P 3	P 4	P 3	291
																																923



CLINICAL CHART, CASE XIV.—William S., aged 19 years; idiopathic epilepsy since 3 years of age. Typical status epilepticus from its first inception. Its fatal ending apparently unmodified by the sedative treatment. A typical relationship between the number of G. M. attacks and the rise in fever, pulse and respiratory curves.

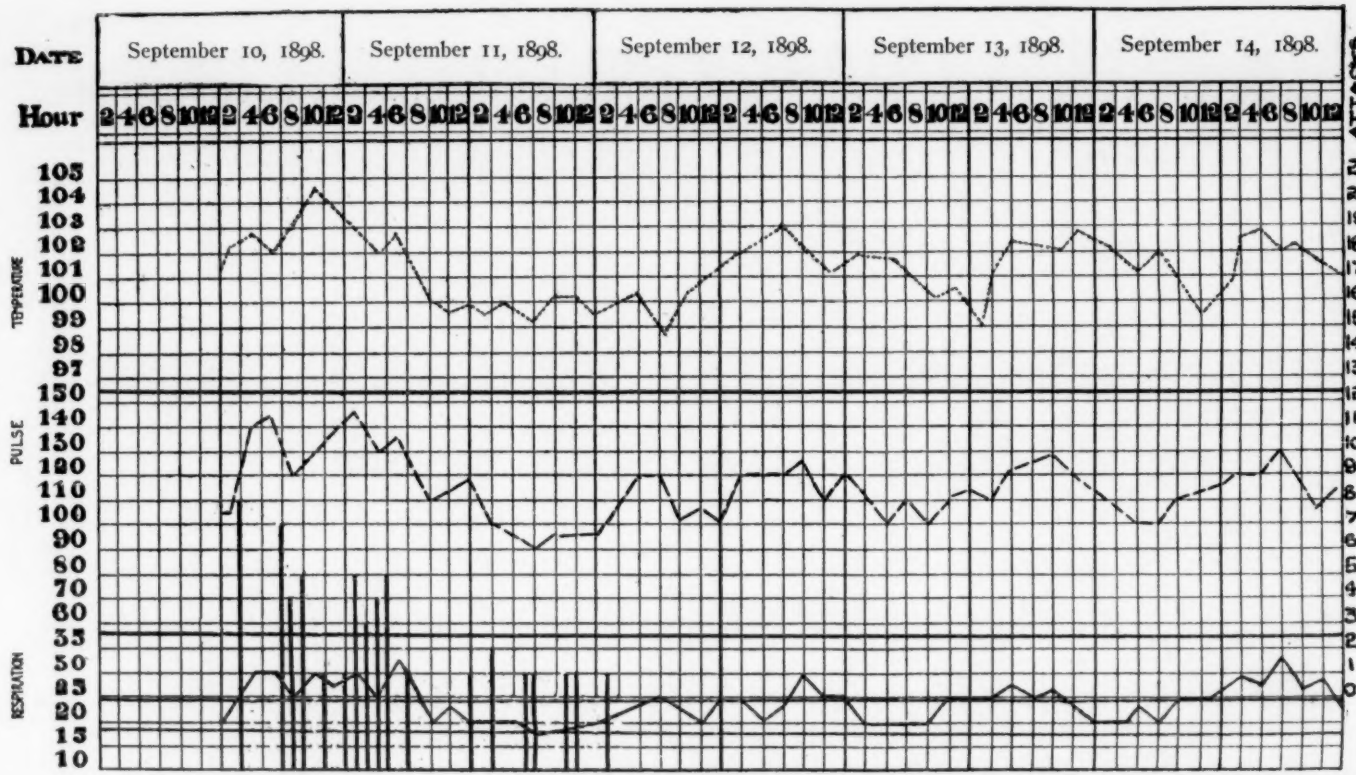


CLINICAL CHART, CASE XV. (a) of John P., aged 24 years, hemiplegic epileptic from 3 years of age. Chart (a) shows but the end of the convulsive period of a status marked (x) in the patient's annual register of seizures which accompanies. There were 137 G. M. attacks with a Jacksonian order of involvement in this period. Note the marked secondary rise in cardinal curves at the reappearance of a few convulsions in the stuporous (exhausted) stage which shows in a graphic manner the error in discontinuing sedative treatment too soon. The cessation of fever and the return of the pulse and respiration to the normal after the stuporous stage are typical.

CLINICAL CHART, CASE XVI. JOHN P., aged 24 years, developing epilepsy from 5 years of age. Chart (a) shows out the end of the convulsive period of a status marked (x) in the patient's annual register of seizures which accompanies. There were 137 G. M. attacks with a Jacksonian order of involvement in this period. Note the marked secondary rise in cardinal curves at the reappearance of a few convulsions in the stuporous (exhausted) stage which shows in a graphic manner the error in discontinuing sedative treatment too soon. The cessation of fever and the return of the pulse and respiration to the normal after the stuporous stage are typical.

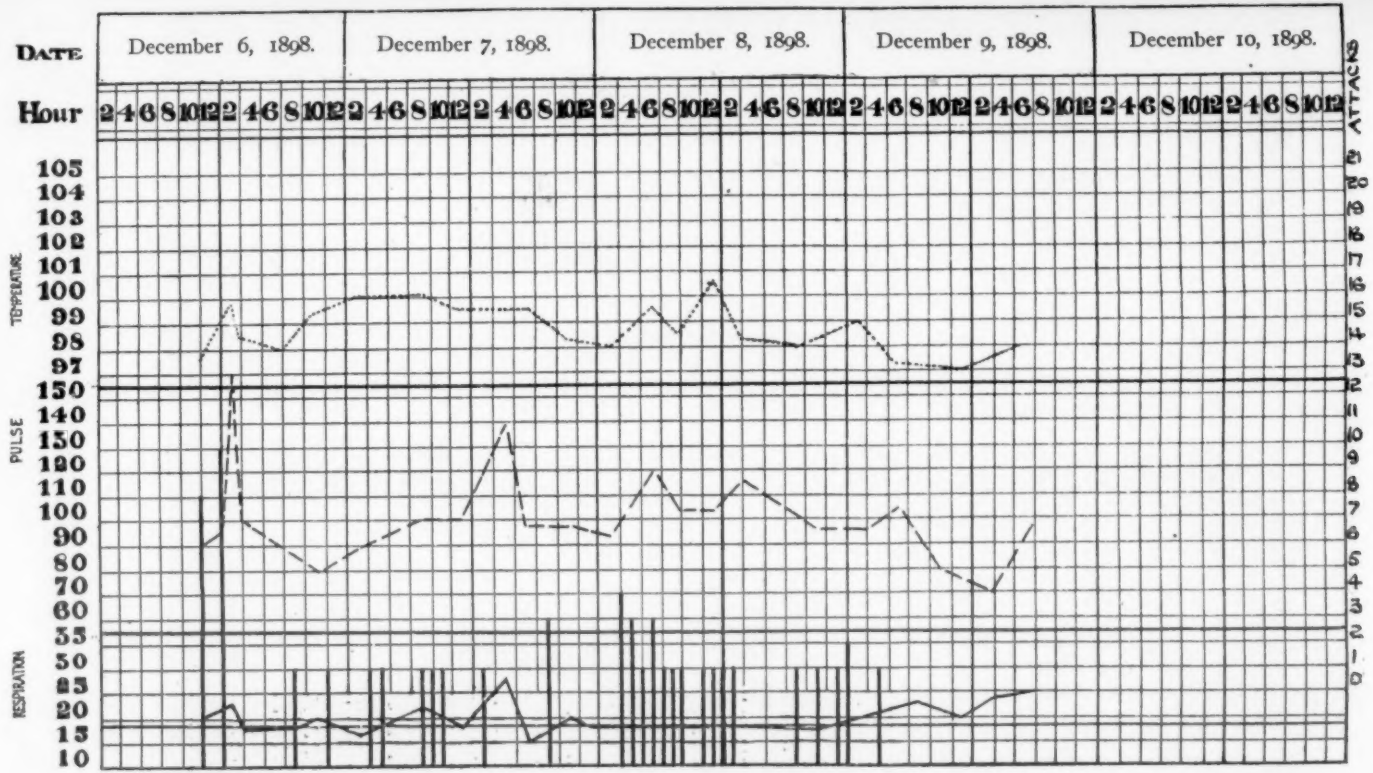
JOHN P.

1898	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	Total.
J...	P 2	P 8	..	..	..	..	G 2	P 1	..	..	..	..	P 5	P 3	..	..	..	..	..	..	P 2	P 2	..	P 2	G 3	P 1	..	..	P 1	P 33	P 3	68
F...	..	P 7	P 3	..	P G 2.2	..	G 1	P G 4.4	P 14	G 14	G 2	P G 6.7	P 20	G P 4.11	G 15	G 10	G 13	G 6	G 8	G 6	G 3	P 1	P 2	P 1	G 1	P 1	P 5	..	..	..	..	167
M..	P 4	P G 1.3	..	P 1	P G 2.1	P 3	..	P 4	..	..	..	G 1	..	..	P 3	P 1	P 1	..	..	P 1	P G 1.2	P 1	..	..	..	P 2	..	..	..	..	P G 2.1	35
A...	..	..	..	P 2	P 1	..	..	P 1	..	P 3	..	..	..	..	G 2	..	..	..	..	P 7	..	P 1	..	..	..	G 2	P G 2.1	..	..	..	..	22
M..	G 2	G 1	..	..	P G 2.4	G 1	..	P G 3.1	..	P 1	..	P 1	G P 2. P 2	G 1	G 1	..	G 1	G 1	G 1	..	..	..	..	P 4	..	P 1	P 1	..	..	P 1	P 2	34
J...	P 1	..	..	P 2	..	..	..	..	..	..	..	G 4	G 13	G 5	⊙	G 33	G 65	G 22	G 7	..	..	..	..	..	P 36	..	..	..	..	..	..	209
J...	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	G 1	..	..	G P 1.1	G 1	..	..	..	..	4
A..	..	..	..	W 1	..	..	P G 1.2	P 1	..	..	..	..	G 1	..	..	..	..	..	..	P 1	..	..	P 3	P G 1.1	..	G 3	P 1	G 2	G 1	..	..	18
S...	G 1	P G 1.1	⊙	G 10	G 3	G 8	G 6	G P 9.2	G 8	G 84	G 26	..	..	..	..	..	P 1	P 2	P 2	..	G. P 4.5	..	..	..	P 1	..	..	G P 8.10	G 12	G 9	..	196
O...	G 22	G 11	G 10	G 10	..	..	..	..	..	P 1	..	P 1	..	P 1	P 8	..	..	..	P 3	G 4	..	..	..	G 1	..	..	..	P 3	P 6	..	..	81
N..	..	P 1	P 1	..	..	..	G 3	P 3	..	..	..	..	..	..	..	..	..	..	..	P 2	P 1	..	G 1	..	..	..	P 1	G P 2	P G 2.4	P G 4.1	⊙	26
D..	P 1	P G 1.15	P 16	G 17	P G 1.14	G P 27.3	G P 16.4	P 6	G P 1.6	P G 2.5	G P 12.1	G 3	G P 12	..	..	P 1	P 1	P 2	P 2	P 1	P 25	G P 3.3	P 15	P 6	G 5	P 2	P 1	P 1	P 4	G 1	P 2	228
																																1088



CLINICAL CHART, CASE XV.—Continued.—(b) The latter part of another convulsive period of status is here shown as it yielded to treatment. Note the secondary rise in the stuporous stage. See annual register for the September period of status marked (x).

treatment. Note the secondary rise in the stuporous stage. See annual register for the September period of status marked (x).



CLINICAL CHART, CASE XV.—Continued.—(c) Third status period aborted by prompt sedation. This patient has had 9 similar status periods in 3 years.

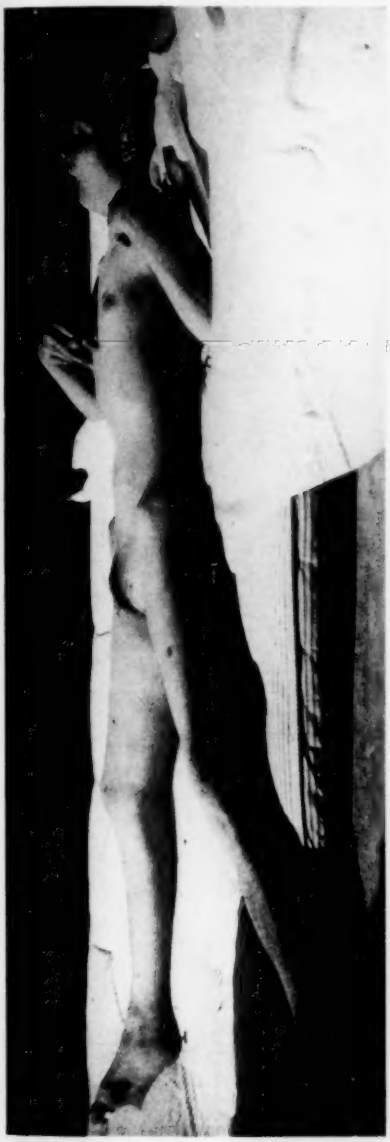


FIG. 1.

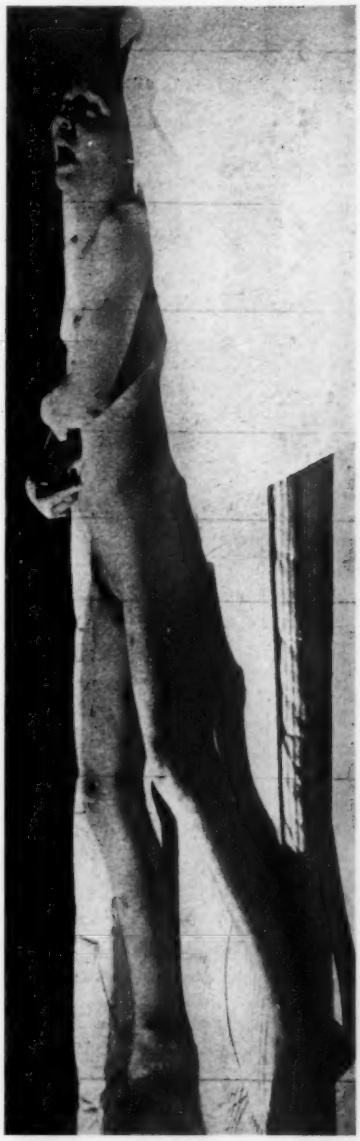
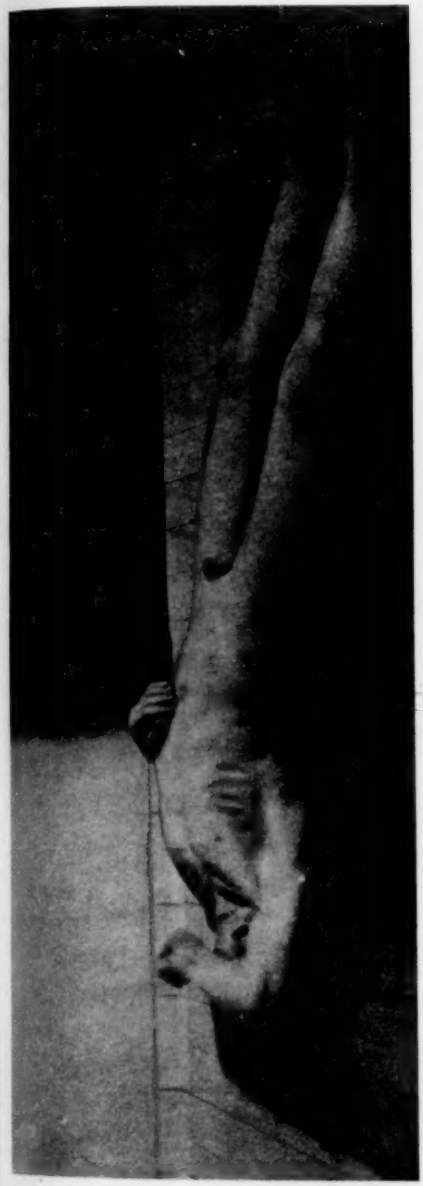


FIG. 2.

Fig. 1. Case IV. Fannie D. (Status Epilepticus). Photograph in second stage of the fit (convulsion in first period involved the left side slightly for a few seconds only). Tonic spasm is just beginning to involve the right face, hand and leg. All muscles of left side are flaccid. The case is one of idiopathic epilepsy with a Jacksonian order of muscular invasion and sequent exhaustion paralysis.

Fig. 2. Photograph of the same case in the third period or stage of the same fit during the secondary and severest involvement of the entire left side—the side first and most engaged in the fit—and followed by exhaustion paralysis. The right hand and foot, although now flaccid, still retain the mold of the second stage of the fit.

Fig. 3. Case IV. Fannie D. Photograph taken two months after the status in February shown in the other two photographs, and two weeks before the fatal status period shown in final clinical chart. The left hand still shows the exhaustion paralysis not yet fully recovered from. No macroscopical evidence of organic lesion at autopsy.



J. K. FIG. 1.

J. K. FIG. 2.

Fig. 6. Joseph K., aged 12 years; infantile spasmodic hemiplegia; frequent periods of status unilateralis in which the period is composed of one continuous imbricated convulsion with varying remittent intensity for two or three hours; the convulsion never entirely ceases during the status period. The first photograph shows hemispasm in the right arm, face and neck, while the second (Fig. 2) shows spasm beginning at the right angle of the mouth just at the moment of muscular relaxation in the arm belonging to the previous fit (Fig. 1). After status of one hour, the temperature in right axilla was 102° F.; left 101.7° F.; rectum 103.1° F. One hour after status had been aborted temperature in right axilla 99.7° F.; left axilla 99.1° F.; rectum 100.8° F.; three thermometers registering alike were simultaneously employed.

Fig. 7. Case, C. P.: a general convulsion in terminal convulsive stage of fatal status in an idiopathic epileptic (clinical chart was typical of acute status). The right arm and left leg are most convulsed; left quadriceps femoris, left calf muscles and all muscles of right arm show this fact clearly. Left arm and hand but slightly involved. Head and eyes are rotated to the left.

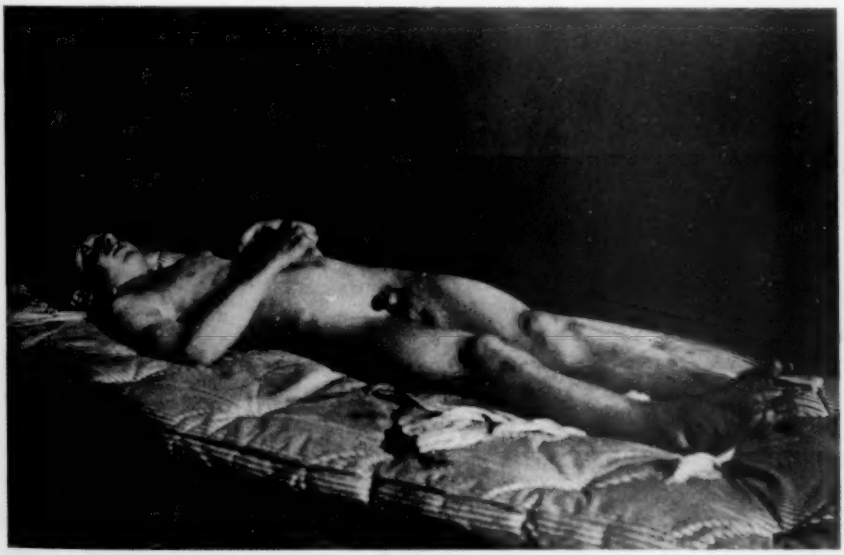


Fig. 8. The second photograph of the same patient in the stertor period immediately after convulsions photographed in the first. The right arm and left leg show contracture, the so-called post-paroxysmal paralytic equivalent. Note the muscle rigidity in biceps and the rigid inversion of the left foot. The contractures ceased a few seconds before the next fit.

SOME METABOLISM STUDIES.

WITH SPECIAL REFERENCE TO MENTAL DISORDERS.[1]

By OTTO FOLIN, Ph. D.,

With the cooperation of PHILIP SHAFFER, B. A., and more recently with the assistance of L. A. HILL, B. A.

Introduction.

Bunge in his text-book of physiological chemistry (4th edition, page 347) and more recently in his text-book of physiology, vol 2, p. 420, points out the curious fact that there is no record in the literature of any one having ever made an analysis of normal urine sufficiently comprehensive to include the determination of all the more important constituents in one and the same sample. On the pages cited Bunge therefore gives the results of two such analyses which he himself had made in connection with some unpublished metabolism studies in order, as he says, to give a picture of the composition of normal urine. G. Donzé and Prof. Lambling call attention to this lack of comprehensiveness in all recorded urine analyses and publish eight such in the March number (1903) of the Journal de physiologie et de pathologie générale.

In addition to these ten analyses should be mentioned three made by Richardson in 1899 at the Mount Hope Retreat and published in the Laboratory Bulletin of that institution. Richardson's analyses unfortunately do not include the kreatinin, probably for lack of sufficient quantity of urine, and probably for the

[1] The chemical laboratory of the McLean Hospital was established on its present basis in the fall of 1900. The investigations pursued during the intervening time have been chiefly concerned with the metabolism of the patients of this hospital, but nearly all the papers published have dealt with technical details and have appeared in other journals.

The work has now reached a stage, however, where it is possible to present a résumé of what has so far been accomplished, and to do so at the present time has seemed desirable to Dr. Edward Cowles, who has continually taken the greatest interest in the work, as embodying a part of a broad plan for the investigation of insanity which he formulated many years ago in his annual reports of the hospital.

same reason do not take the metals into consideration. The analyses are included in an article on the secretions in general and are published without any comments either as to analytical methods used or as to the significance of the results obtained, but must nevertheless be cited here as a very creditable attempt to make urine analyses as complete as possible.

There may, of course, be a few other similar analyses scattered through the literature, but as far as I know those mentioned are the most complete on record.

As a matter of fact it is here to be noted that neither the analyses of Bunge nor those of Donzé and Lambling can be considered very complete. Bunge determined all the more important inorganic constituents evidently for the purpose of determining whether the acid or the basic elements were there in preponderance. In such a study he certainly should also have determined the ammonia which he, as he himself regretfully remarks, neglected to do. A far more serious, in fact an almost unaccountable oversight on his part, is the fact that he also neglected to determine the total nitrogen. Without this factor Bunge's other determinations (urea, uric acid, kreatinin) can be given very little value as an index to the normal distribution of the nitrogenous compounds of urine.

The analyses of Donzé and Lambling are practically complete with respect to the nitrogenous constituents of urine and, as the first of the kind ever published, they are exceedingly interesting and must be accorded due credit.[2] But these analyses do not present a composite picture of normal urine in its entirety because Donzé and Lambling left out of their determinations all the inorganic constituents. In this respect they confined themselves to determinations of the total mineral residue obtained

[2] The analyses of Donzé and Lambling are further important in connection with their estimation of the total weight of undetermined organic materials occurring in urine and usually classified under the meaningless term "extractives." Donzé and Lambling find these substances to be present in urine in much larger quantities than is usually supposed. It may, however, fairly be questioned whether a mixture of inorganic salts such as are found in urine can be deprived of all water of crystallization by means of a vacuum and sulphuric acid at ordinary temperatures and whether the results obtained by Donzé and Lambling are not therefore too high. Calcium chloride and magnesium chloride, for instance, would certainly be present and would retain water under such conditions.

after igniting the "total solids" according to the method of Neubauer and Vogel.[3] This laborious process is, however, as I indicated in my paper on the acidity of urine[4] open to sources of errors that make it very inaccurate. In regard to the inorganic constituents of urine the investigations of Donzé and Lambling cannot therefore be said to have contributed anything of value.

As far as I am aware, it is consequently in accordance with the facts and doing injustice to no one to state that there is still no record in the literature of a single complete analysis of one and the same twenty-four hour quantity of urine—complete in the sense of including all the more important substances that are frequently determined singly or in small groups.

The reason for this state of affairs is not far to seek. The substances present in sufficiently large amounts to be of importance in a discussion of the composition of urine are quite numerous. The available methods for the determination of several of these substances are not only far from reliable, but they consume a great deal of time, and they require in addition so much urine as almost to exclude the determination of all in one normal twenty-four hour quantity, especially as no one would care to undertake such a series of determinations without having enough material on hand for at least a few duplicates.

It might fairly be asked what would be the use or advantage of laboriously trying to determine all the more important constituents in one 24-hour quantity of urine as against the current practice of determining simply this or that or a few constituents which may seem of special interest in the course of any particular investigation? In regard to a few isolated urine analyses it must be admitted that the advantage gained may not be very great, although, it would certainly be desirable to have an easily accessible record of at least a few such analyses in order to give a more definite picture of the composition of normal urine.[5]

[3] Analyse des Harns, 10th edition, 1898, p. 703.

[4] American Journal of Physiology, vol. 9, p. 273 (1903).

[5] It would certainly be a point of advantage if, by means of a few such records, it would be possible to check, at least in some measure, the ridiculously loose usage in medical literature of the terms "normal," "increased," "diminished" as applied to the various urinary constituents—terms which seem intended to imply that quantitative determinations have been made when such is not the case.

In connection with long continued, carefully planned metabolism experiments, it must further be considered of the utmost importance to determine the greatest possible number of factors represented in the urine. Every additional determination has then an increasing value not only because of the increasing completeness with which the results represent the urine as a whole, but also and more because of the way in which each such factor helps to detect whatever there may be of errors in the determinations or of anomalies in the excretion. There can scarcely be any doubt that a great deal is yet to be learned in regard to the laws of the urinary excretion and of animal metabolism by a sufficiently complete system of analysis which would record many peculiar, unexplained variations from day to day in the several urinary constituents, variations which are sometimes exceedingly pronounced even under uniform conditions of diet and occupation.

Another important reason why metabolism experiments should be accompanied by the completest possible urine analyses is the broadened foundation which they would give for similar studies of certain diseases, such as, for example, diabetes, rheumatism, and gout, which are admittedly accompanied by or due to one or another metabolism disorder, and which moreover lend themselves almost as well as normal cases to just such studies.

Among the diseases pre-eminently accessible to metabolism studies must be classed the mental disorders. Whatever may be one's estimate of the vague and indefinite ideas associated with such terms as auto-intoxication, deranged nutrition, and abnormal metabolism in connection with the more general forms of insanity, the numerous publications on these subjects furnish abundant evidence of the existence of a widespread hope that chemical investigations will, perhaps, sooner or later yield a clearer understanding of these diseases than can be looked for from the other sciences that have been brought to bear on them. Such a hope may be wholly illusionary. Abnormal metabolic processes must unquestionably play a more or less important part among the pathological conditions that produce various forms of insanity, but it may well be that the chemical methods at present available are entirely too crude for the detection or study of these deviations from the normal.

It seems unnecessary to discuss here how much or how little advance in the study of insanity is to be expected from chemical methods of investigation. I do, however, wish to emphasize the fact that the road of inquiry along the line of metabolism experiments is open, definite, and clear-cut, and that it should not be particularly difficult to plan such experiments comprehensive enough and extensive enough to yield all the information that the present state of chemical technique can yield along this line. The chief difficulties naturally encountered in such investigations have already been mentioned and indicate the limitations of the inquiry from an analytical standpoint. Other difficulties such as those connected with the untidiness and violence of the very patients who would probably show the most instructive results indicate the probable limitations from another standpoint. Within these limitations the field of inquiry is, however, so clear as to demand imperatively more systematic experiments.

Notwithstanding the difficulties and labor involved in making far more complete urine analyses than are yet on record, it would seem as though such work ought to be done to the fullest possible extent—at least such were the conclusions reached by the writer when he, in the fall of 1900, was called by Dr. Cowles to the McLean Hospital to study insanity from a chemical standpoint, and more particularly to make investigations into the nutrition of the patients of this hospital.

The incompleteness of all metabolism experiments of the past as judged by the comprehensiveness of the accompanying urine analyses is particularly pronounced in the metabolism records of the mental diseases. A review of this literature was published by A. Schaefer in the Monatsschrift für Psychiatrie und Neurologie (1897), Nos. 2, 3, 5, and 6. In regard to these investigations says Schaefer, "it must first of all be emphasized that they have all one thing in common, namely, that none is concerned with the metabolism as a whole, i. e. with all the processes involved in the intake and outgo, in the breaking down and the building of the chemical products that constitute the body-substance." The following divisions of the subject are treated in these investigations (about 267 in number), the number of papers devoted to each division being indicated in the list below:

Gain or loss of weight	27	papers.
Albuminous substances in the urine	46	"
Glycosuria	28	"
Acetonuria	12	"
Toxines in the urine	9	"
Uric acid in the urine	15	"
Kreatinin in the urine	8	"
Oxalic acid in the urine	7	"
Chlorides in the urine	10	"
Phosphates in the urine	42	"
Sulphates in the urine	9	"

The fragmentary nature of all metabolism papers published up to the year 1897 is equally common to those which have appeared since, nor is the accuracy of the experiments or the validity of the conclusions reached any less open to question.

Analytical Technique.

The difficulties involved in the carrying out of a more comprehensive scheme of urine analysis seemed, perhaps, greater than usual about the time that this work was begun. The best available analytical technique, especially the methods for determining the more important nitrogenous constituents, had been shown to be less reliable than was formerly supposed, and new methods or modifications offered as substitutes, either made the determinations even more time-robbing and cumbersome than before or did not seem to offer any greater assurance of being reliable.

See for example Braunstein,[6] Salaskin and Zaleski[7] on the determination of urea, Wörner[8] and Jolles[9] on the determination of uric acid, Salkowski[10] on the determination of kreatinin, and finally Nencki and Zaleski[11] and Camerer and Söldner[12] on the determination of ammonia.

The fact that the metabolism work to be undertaken would probably be one of long duration taken together with the uncertainty or lack of adaptability of several of the most important analytical methods must here be cited in explanation of the plan

[6] Zeitschrift f. Physiol. Chemie, vol. 28, p. 73.
[7] The same, vol. 31, p. 473.
[8] The same, vol. 29, p. 70.
[9] The same, vol. 29, p. 222.
[10] The same, vol. 14, p. 471.
[11] The same, vol. 33, p. 193.
[12] Zeitschrift f. Biologie, vol. 38, p. 236.

that has been pursued in connection with these studies. It seemed necessary to subject the analytical methods to be used to a critical examination so as to know from personal experience their limits of accuracy and the sources of possible errors. It also seemed worth while to try to modify some of the current methods and to devise some new ones in order to obtain finally a scheme of routine analysis as accurate as possible and rapid enough for daily use—a necessity in long continued feeding experiments.[13]

It would be out of place in a paper of this kind to go into the details of such preliminary investigations of analytical methods. Since, however, the validity of the findings must to a very great extent depend upon the accuracy of these methods, a brief summary of our work in this field, giving at the same time a description of each method as now used, would seem to be necessary.

1. *Urea.*—A new method for the determination of this urinary constituent was devised at the very beginning of the work. The method was first published in Zeitschrift für Physiol. Chemie, vol. 32, p. 504. Within a relatively short time the work was repeated in Hannover by Prof. Arnold and C. Mentzel who published an exceedingly unfavorable report of the method in the same journal, vol. 36, p. 48. The question of urea determinations was again taken up and it was shown that the objections raised by Arnold and Mentzel were unwarranted by the facts (see the same journal, vol. 36, p. 337, and vol. 37, p. 548).

In the meantime the method had been subjected to a remarkably thorough and severe examination by Prof. K. A. H. Mörner, joint author of the well-known Mörner-Sjöquist method for determining urea.[14] Since Mörner's numerous experiments corroborate in every detail all that has been claimed for this method, it would seem safe to assume that its accuracy is now established. (This view is further justified by the failure of Arnold and Mentzel to attempt any defense of their unfavorable report, and

[13] The so-called clinical methods such as, for example, the hypobromite methods for determining urea or the various centrifuge methods of Purdy and of Poehl, methods which may be rapid enough, but which from the nature of the case cannot be accurate, have not been considered in these investigations.

[14] Skandinavischen Archiv f. Physiologie, vol. 14, p. 297.

by the fact that the method has been tested by Donzé and Lambling loc. cit. and by Sallerin—Journal de physiologie, etc. 1903, p. 259—who have also reported in favor of the method.) Mörner introduced a few minor changes in the practical execution of the urea determinations. In regard to these it must be said that they were undoubtedly quite suitable for his purpose, a critical examination of the method, in connection with which the fact of lessened expediency was of no importance. Mörner's paper was, moreover, sent to the printer before the appearance of my second paper on the subject in which similar, but more convenient, modifications were described.

Mörner further reported one important discovery, namely, that in sugar urines the method does not give reliable results and that to get accurate values for such urines a combination of my method with the Mörner-Sjöquist method, slightly modified for the purpose, is necessary. It may here be added that diabetic urines have not yet occurred in our metabolism experiments, but that we have nevertheless used this combination of the two methods in quite a few urines, as was indeed the original intention which was abandoned only because equally good results were obtained without it.

The method is used in this laboratory as follows: 5 cc. of urine are measured into an erlenmeyer flask (capacity 200 cc.), 5 cc. con. hydrochloric acid, 20 g. crystallized magnesium chloride, a piece of paraffine the size of a small hazelnut, and finally two or three drops aqueous 1 per cent solution of "alizarin red" are added. The safety tube described in my second paper on the subject is then inserted and the mixture boiled on an electric oven until each returning drop from the safety tube produces a very perceptible bump. The heat of the oven is then reduced somewhat and the heating is continued for a full hour.

Particular attention must be called to the addition of the indicator alizarin red, as its use in this combination has not been described before. This indicator is so stable as to withstand the heat and it turns bright red in the presence of the smallest trace of alkali. The boiling contents in the erlenmeyer flask must not be allowed to remain alkaline and as soon as they begin to turn red a few drops (but only a few) of the acid distillate in the safety tube are shaken back into the flask. At the end of an hour the contents of the flask are put into a liter flask with about 700 cc. water, and 20 cc. 10 per cent sodic hydrate and the ammonia distilled off. This distillation must be continued until the contents of the liter flask are nearly dry or till the distillate shows no trace of ammonia with delicate litmus paper. The distillate is then boiled a few minutes to drive off the car-

bonic acid, cooled and titrated in the usual manner. The ammonia of the urine as well as that of 20 g. magnesium chloride is subtracted from the result obtained.

With the electric ovens in our laboratory we usually carry out from two to four urea determinations simultaneously.

2. Uric Acid.—My modification [15] of Hopkins' method for determining uric acid has, on account of its simplicity, come into somewhat general use. In its original form this method was found to be open to question on the point of accuracy (Wörner, Jolles, loc. cit.). To rectify these sources of error proved by no means an easy task, but it was finally accomplished and the method as described by Shaffer and myself in the above mentioned journal, vol. 32, p. 552 (1901) gives undoubtedly, at least in our hands, as accurate results as can be obtained by the far more tedious methods of Ludwig-Salkowski, or Hopkins. Our work has since been repeated by Fr. Hupfer [16] and by Donzé and Lambling (loc. cit.) who found the method satisfactory. A rather more qualified approval has come from C. P. Emerson (Johns Hopkins Hospital Bulletin, January, 1903) who, on the basis of results obtained by students, pronounces the method the best of the permanganate methods, but still by no means an easy determination. Emerson curiously enough finds the first description of the method fully as satisfactory as the last and also thinks that the reagents used must be of exceptional purity. This is not in accordance with our experience.

The uric acid determinations are carried out in our laboratory as follows:

The chief reagent is a solution of 500 g. ammonium sulphate, 5 g. uranium acetate and 60 cc. 10 per cent acetic acid in 650 cc. water.

150 cc. urine is measured into a tall, narrow beaker, or a cylinder, and 37½ cc. of the reagent is added (if the available quantity of urine is not too small, 200 cc. urine and 50 cc. reagent are used). The mixture is allowed to stand without stirring for about half an hour. The uranium precipitate has then settled and the clear supernatant liquid is removed by siphoning or by decantation. 125 cc. of this liquid is measured into another beaker, 5 cc. strong ammonia is added, and the mixture set aside until the following day. The precipitate is then filtered off, washed with 10 per cent ammonium-sulphate solution until the filtrate is quite or nearly free from

[15] Zeitschrift für Physiol. Chemie, 1898, vol. 24, p. 224.

[16] Zeitschrift für Physiol. Chemie, vol. 37, p. 302.

chlorides. The filter is removed from the funnel, opened and the precipitate rinsed back into the beaker. Enough water to make about 100 cc. is added and finally the precipitate is dissolved by means of 15 cc. con. sulphuric acid and at once titrated with $\frac{n}{20}$ potassium permanganate solution, each cc. of which corresponds to 3.75 mg. uric acid. A correction of 3 mg., due to the solubility of the ammonium urate, is added to the result. The very first pink coloration, extending through the entire liquid from the addition of two drops permanganate solution while stirring with a glass rod marks the end point of the titration.

3. Ammonia.—Up to a few years ago the determination of ammonia in urine received but little critical attention and so it happened that unsuitable modifications gradually crept into the text-book directions for determining this substance according to Schlösing, the only method in use.

In connection with these researches more time and attention has been given to a study of this determination than to that of any other constituent. Three methods for determining it have been published. The first method, though very briefly described in Zeitschrift für Physiol. Chemie, vol. 32, p. 515, has found its way into several text-books and has met with some favorable comment in the literature (see Sallerin, Journal de physiologie et de pathologie générale).

Further investigations led, however, to the discovery that neither this method nor the Schlösing method could be considered reliable (see Shaffer: American Journal of Physiology, vol 8, p. 330).

The subject was accordingly taken up again and two other methods were devised, one by Shaffer and one by myself. Shaffer's method, the principle of which is a vacuum distillation in the presence of alcohol, gives undoubtedly very satisfactory results. This method seems to have been discovered almost simultaneously by Krüger, whose publication did, however, not appear till six months later than the publication from this laboratory. (See Zeitschrift für Physiol. Chemie, vol. 39, p. 165, and also p. 477).

My own method is based on the principle of driving off the preformed ammonia in the urine by means of an air current, and as this method possesses the advantages of simplicity together with absolute accuracy it has now for about a year been used exclusively in connection with our metabolism experiments.

This method will therefore be briefly described here. A good water vacuum pump, such as is used in every laboratory for vacuum distillations, or better still a similarly arranged force pump, must be used to furnish a sufficiently rapid air current.

25 cc. is measured into a tall aërometer cylinder which is filled with a double-bored rubber stopper, through which passes one long and one short glass tube exactly as in an ordinary so-called washing bottle, the longer tube reaching almost to the bottom of the cylinder. The shorter tube consists, however, of a calcium chloride tube and is filled with cotton.

About one gram sodic carbonate and about 5 cc. petroleum are added to the urine in the cylinder. The upper end of the calcium chloride tube is connected with another glass tube[17] which leads to the bottom of a wide-mouth 16-oz. bottle half filled with water and 25 cc. $\frac{n}{10}$ acid and some indicator.

A strong air current is then passed for an hour and a half through the alkaline urine and the bottle containing acid, and carries with it every trace of ammonia contained in the urine.

This ammonia is equally completely absorbed by the acid and then is determined by direct titration.

The air coming from the acid where the ammonia is absorbed is then led through a second similar apparatus, thus enabling us to make two ammonia determinations at the same time with an accuracy for all kinds of urine or other animal fluids which I do not hesitate to say is not to be equalled by any other known method.

4. Kreatinin.—No text-book with which I am familiar gives any adequate understanding of the difficulties involved in the determination of kreatinin in urine. The larger books usually give the several methods proposed without comment, and the smaller ones give one or none, likewise without comment.

Neubauer's method or Salkowski's modification of the same is, as a matter of fact, the only one which has been used at all generally in the few kreatinin determinations scattered through the literature, and that this method is in no sense reliable or accurate is clearly to be gathered from Salkowski's last paper on the subject (Zeitschrift für Physiol. Chemie, vol. 14, 1890, p. 471).

The operations involved in this determination of kreatinin are moreover among the most tedious and time-robbing that we have

[17] This tube can also to advantage be of special construction to insure more perfect absorption of the ammonia; such a tube is described in the original paper on the method and is now made for us by Eimer and Amend in New York.

in connection with the determination of any urinary constituent. In consideration of these facts and of the further fact that each determination requires from two to three hundred cubic centimeters of urine, i. e. in some cases a third of a whole 24-hour quantity, it seemed wholly impracticable to try to include kreatinin determinations in the work unless a more suitable method could be found.

An attempt was therefore made to devise a new method which would enable us to include kreatinin determinations. A colorimetric method requiring but 10 cc. of urine for each determination, and like all colorimetric methods extremely rapid in execution, was successfully worked out. It is believed to be reasonably accurate. As this method has not yet been published, although it has been in constant use for almost a year, it will be described here a little more in detail than any of the preceding methods.

The principle of the method is the color reaction which kreatinin gives with alkaline picric acid solution. According to Jaffé, the discoverer of this reaction, it is characteristic for kreatinin and is given by no other normal urinary constituent. The only substances which I have found to give the reaction under the conditions of the experiment are aceto-acetic ether, acetone and hydrogen-sulphide. Since these can all be removed from the small quantity of urine used in this determination by a few minutes heating with dil. hydrochloric acid and are moreover rarely present, this fact is of but little consequence. A fortunate circumstance discovered in the course of the investigation is the fact that the color produced is identical in suitable concentration with the color of potassium bichromate solutions. This gave a standard for comparison as easy to prepare as it is stable. The half normal solution was selected for the purpose.

In order to make color comparisons accurate enough for quantitative purposes a high-grade colorimeter was necessary and the French instrument of Duboscq was secured from Eimer and Amend "on approval." This instrument proved eminently satisfactory. The construction and workmanship are excellent, the prisms are absolutely flawless and the height of each solution can easily be adjusted to within an accuracy of a tenth of a millimeter.

Perfectly pure kreatinin was then prepared and the conditions under which its highest colorimetric value is produced was ascertained, and this value was determined in terms of the standard bichromate solution. It was found that 10 mg. kreatinin dissolved in about 10 cc. water gave the highest value five minutes after the addition of 15 cc. saturated picric acid

solution and 5 cc. 10 per cent sodic hydrate. The resulting solution diluted at the end of that time to 500 cc. showed about the same color as the $\frac{2}{n}$ bichromate solution when viewed in the colorimeter (i. e. by transmitted light) through a depth of 8 mm. To be more accurate 8.1 mm. of the kreatinin picrate solution was exactly equal to 8 mm. of the bichromate.

The absorption of light was further found to be quite different for the two solutions, but this fact can not be discussed here except in so far as to state that this necessitated the making of a definite depth of the bichromate the standard for comparison. 8 millimeters was chosen for the purpose.

On the basis of these facts the kreatinin determination in urine is made as follows:

Half normal potassium bichromate containing 24.55 g. per liter; saturated picric acid solution containing about 12 g. per liter; and 10 per cent sodic hydrate solution, are the reagents needed.

10 cc. urine are measured into a 500 cc. volumetric flask, 15 cc. picric acid and 5 cc. sodic hydrate are then added and the mixture is allowed to stand for 5 to 6 minutes. This interval is used to pour a little of the bichromate solution into each of the two cylinders of the colorimeter. The depth of the solution in one of the cylinders is then accurately adjusted to the 8 mm. mark. With the solution in the other cylinder is made a few preliminary colorimetric readings, simply for the sake of insuring greater accuracy in the subsequent readings of the unknown solution. The two bichromate solutions must of course be equal in color, and in taking their readings no two should differ more than 12 mm. from the true value (8 mm.), leaving out of consideration the very first reading made, which is sometimes less accurate. Four or more readings should be made in each case and an average taken of all but the first. After a while one becomes very sure of the true point and can take the average of the first two readings.

At the end of five minutes the contents in the 500 cc. flask are diluted up to the 500 cc. mark. The bichromate solution is thoroughly rinsed out of one of the cylinders by means of the unknown solution and several colorimetric readings are then made *at once*.

The calculation of the results is very simple. If for example it is found that it takes 9.5 mm. of the unknown urine picrate solution to equal the 8 mm. of the bichromate, then the 10 cc. of urine contains $10 \times \frac{8.1}{9.5} = 8.4 +$ mg. kreatinin.

The amount of urine taken for the determination is usually 10 cc., but if this should be found to contain more than 15 or less than 5 mg. kreatinin the determination should be repeated with a correspondingly different amount of urine, because outside of these limits the determination is much less accurate. With kreatinin solutions the results are uniformly surprisingly accurate, and I have as yet found no reason for believing that it is not equally reliable, at least for normal urines. The color of the

urine does not materially affect the result on account of the very great dilution.

A kreatinin determination can be made by this method in less than 15 minutes.

The preceding four methods for the determination of the nitrogenous constituents have been described somewhat in detail. The remaining analytical methods, although also representing some new technique devised in connection with these metabolism studies, can be passed over more briefly.

5. *Specific Gravity.*—The ordinary urinometers used for determining the specific gravity of urine were very early found to be unreliable, and this fact was reported in the paper on Phosphate Metabolism (American Journal of Physiology). After failing to find a single one bought in the open market correct between the values 1.015 and 1.025, we had some special ones made to order and these were found satisfactory. With these the specific gravity has been taken in the ordinary manner.

6. *Volume.*—Measured in ordinary cylinders.

7. *Chlorides.*—The chlorides were titrated according to Volhard, and the only point that need be mentioned is the fact that a fair amount of nitric acid must be added in order to get a clear filtrate for the titration of the excess of silver nitrate.

8. *Total Sulphates.*—The total sulphates were determined gravimetrically according to method described by the writer in the American Journal of Physiology (vol. 7, p. 152).

For awhile even after this method had been published it was thought that the chlorate added to destroy the coloring matter might be the source of some error owing to the possible oxidation of some "neutral" sulphur. As more and more analytical data have accumulated this fear has been shown to be unnecessary, because if this were so to any noticeable extent it would make the ethereal sulphates abnormally high, as they are determined according to the same principle, and this is not the case.

In connection with the sulphate determination it may be added that the volumetric methods for determining the same were tested and were found unreliable. The complete precipitation of barium-sulphate is in no sense instantaneous enough for a volumetric titration and is in fact never complete except in the presence of a considerable excess of the precipitant.

9. *Ethereal Sulphates.*—The determination of the ethereal sulphates as made in these investigations has never been described, but a few words will suffice for the purpose.

To 200 cc. urine are added 100 cc. 10% barium chloride solution and the mixture is allowed to stand over night at ordinary room temperature. The supernatant liquid is then decanted into a dry beaker and if not absolutely clear is filtered. 150 cc. of the clear filtrate, corresponding to 100 cc. of urine, is then transferred to an erlenmeyer flask and boiled with a few cc. strong hydrochloric acid and enough potassium chlorate to give

a colorless solution. The remaining operations are identical with those described for the determination of the total sulphates (loc. cit.).

10. Neutral Sulphur.—The so-called neutral sulphur is represented by the difference between the total sulphate and total sulphur found in connection with the mineral acidity determinations. In regard to the nature of the substances representing this sulphur practically nothing is known.

11. Phosphates.—The phosphates are determined volumetrically by means of uranium acetate solution, using powdered potassium ferrocyanide as indicator. Chemically pure crystallized monopotassium-phosphate is used to standardize the uranium salt solutions. (See Sutton's Volumetric Analysis, 8th ed., p. 316.)

In this connection it must be added that the so-called organic phosphates have been repeatedly sought for, but have never been found, except in such small traces as to suggest that even these traces represent only remnants of ordinary phosphates which escape precipitation on the addition of calcium chloride and ammonia according to the method of Oertel (Zeitschrift f. Physiol. Chemie, vol. 26, p. 125).

12. Acidity.—Total, mineral, organic.

These acidity determinations are described in my paper on the Acidity of Urine (American Journal of Physiology, 1903, vol. ix, p. 265).

Special attention might be called to the determination of the total acidity of urine. All titration methods for determining this factor have been in recent years quite generally discarded because it was shown that acid phosphates cannot be titrated in the presence of calcium salts. In the above mentioned paper is shown that the disturbing effects of the calcium salts are completely eliminated by the addition of neutral potassium oxalate. It is believed that the method as described, consisting of the addition of about 15 g. oxalate to 25 cc. of urine and titrating immediately, i. e., while still cold from the effect of the dissolved oxalate, with phenalphthalein as indicator, gives the true acidity of urine with greater accuracy than any other known method.

In order to find the relative preponderance of inorganic acids or bases in urine it would be necessary to determine besides the phosphates, sulphates and chlorides, the metals sodium, potassium, calcium and magnesium. This Bunge did in his two analyses. To make all these determinations in addition to the others in a large number of urines would be an enormous task even if the quantity of urine permitted it, which in many cases it would not do. The subject is, however, important in connection with the question of the acidity of urine and the possible occurrence of unknown bases, "preliminary reports" of which are not lacking in the literature. In my paper on the acidity of the urine cited above is described a new method by means of which the preponderance of the inorganic acids or bases can be determined without separate determinations of each of the metals. As I have nothing to add to the details there given, the method will not be described here. The results of these determinations are recorded as "mineral acidity" because in all cases but one the acids are in preponderance.

Attention is here called to the fact that from the mineral acidity can also be calculated how much of the phosphoric acid is present as primary and how much as secondary phosphate.

13. Indican.—The indican determinations recorded in this paper will need a somewhat fuller explanation because the method used for recording these values is wholly arbitrary and has not been described elsewhere. Exactly one-hundredth of a 24-hour quantity of urine is taken for each determination. In this the indigo is developed by the addition of Obermeyer's reagent and the indigo blue is taken out by means of 5 cc. chloroform. With the chloroform solutions are then made colorimetric comparisons, using Fehling's solution as a standard. The Fehling's solution is given the arbitrary value of 100.

The advantage of this method is that it takes very little time and the standard for comparison is one that is kept on hand in every laboratory.

It has been our intention to work out an exact method on this principle, by using the same colorimeter as is used in the kreatinin determinations for the color comparisons and then finding the value of Fehling's solution in terms of pure indigo. The difficulties in the way of such a colorimetric method are by no means small, the chief of which is to produce a pure blue solution from the indican without any admixture of red coloring matter.

The method as described gives sufficiently definite values to enable any one to repeat the experiment and get comparable values, but it does, of course, in the present form not possess the accuracy that would entitle it to be classed as quantitative.

14. Total Nitrogen.—The total nitrogen determination would need no mention were it not for the discussion which the Kjeldahl nitrogen determination has lately received in the current literature. In these experiments the Kjeldahl oxidation was made with the help of a mixture of copper sulphate, potassium sulphate and sulphuric acid. The heating was continued about half an hour, and scores of duplicates which have been made in the course of these investigations have uniformly shown the method to give the true nitrogen value of the urine. The urines for the nitrogen determinations are measured out by means of carefully standarized 5 cc. burettes graduated in twentieths of a cc. The normal acid and alkali were prepared with the utmost care, about 8 liters at a time, with sodium bicarbonate prepared from sodium as control. The pipettes used in making the tenth normal solutions from the normal as well as all burettes used in this work were carefully standardized.

Duplicates.—In connection with the description of the various analytical methods used in this investigation the question of duplicate analyses must be mentioned. The large number of determinations made in each day's urine have made it impossible to include the making of duplicates unless there was reason to think that some error might have crept into the work. It is,

however, quite remarkable how easily errors of determination are discovered in urines obtained from a uniform diet when all the above mentioned constituents are included in the work, and the results are recorded on tables showing the various ratios and percentages. The study of these ratios and percentages has led to numerous duplicate analyses which in turn have led to the discovery of not a few errors to be sure, but in the majority of cases have only served to establish the fact that there is not a single "normal" relationship among the urinary constituents that is not occasionally broken into by unaccountable variations. This will be referred to again.

From what has been said on the preceding pages it will be seen that the urine analyses recorded in this paper are more than usually complete.

Bunge's determinations (loc. cit.) included urea, kreatinin, uric acid; the metals Na, K, Ca, Mg; and the acid constituents Cl, SO_3, P_2O_5. The analyses of Donzé and Lambling included total nitrogen, urea, uric acid, kreatinin, ammonia and the xanthin bases. The determinations made in the course of these researches include, as regards the nitrogenous constituents, total nitrogen, urea, ammonia, kreatinin, and uric acid. The sum of the four last named constituents amounts to from 92 to 98 per cent of the total nitrogen. The remainder of the total nitrogen is represented by a great variety of substances, all present, as far as is known, in so small amounts as to be undeterminable by any practical methods. The xanthin bases of Donzé and Lambling were found for example to amount to only .1% of the total nitrogen. We have determined the xanthin bases in about thirty urines and have obtained about the same amount (.1%), although our urines should have contained less as compared with those of Donzé and Lambling, because the similar product, uric acid, is present in our urines in only about half the quantity found by the two French investigators. Our determinations account, therefore, definitely for 95 or 96 per cent on the average of the total nitrogen eliminated through the kidneys, and are more complete in this respect than any analyses recorded in the literature, with the single exception of those made by Donzé and Lambling.

Besides determining the nitrogenous constituents, we have determined the chlorides, sulphates and phosphates, and in this con-

nection have paid special attention to the phosphate elimination. A few additional determinations have been included, to which neither Bunge nor Donzé and Lambling gave any consideration, but which are certainly of importance in the study of both normal and pathological urines. These determinations include ethereal sulphates, "neutral sulphur," the acidities and indican.

To recapitulate: the quantitative determination included in our urine analyses are as follows: volume, specific gravity, total nitrogen, urea, ammonia, kreatinin, uric acid, chlorides, phosphates, ordinary sulphates, ethereal sulphates, neutral sulphur, total acidity, mineral acidity (or alkalinity), indican.

Diet.

The "complete" urine analyses enumerated in the beginning of this paper were made without reference to the diet from which those urines were obtained, except for the distinction which Bunge made in his analyses between "animal" and "vegetable" diet. The value of such analyses would of course be much greater if the diets of the cases studied were so regulated as to insure comparable values in the urine.

It would seem to the writer that this relation of the food to the products excreted in the urine has not yet received all the attention that it deserves. It is common enough in metabolism experiments to analyze the food and to see to it that the food remains fairly constant during any one series of experiments. This is, however, not enough. Recent investigations, particularly those of Osborne of the Connecticut Experiment Station at New Haven, have shown that different proteins differ far more than was formerly supposed in their chemical constitution. The important practical conclusion to be drawn from this discovery is that in order to get comparable results from *different* metabolism experiments made at different times or by different investigators, the same *kind* of food must be used. The practice of simply providing a known amount of nitrogen together with the necessary "calories" is in no sense adequate for comparative results. That this is so would probably have been discovered long ago if the metabolism experiments had been accompanied by more complete urine analyses.[18]

[18] The fact that different proteins contain different percentages of sulphur and of phosphorus has of course been known a long time, and it is well

If a few "standard diets" could be adopted and come into general use in the same way that "test meals" are used in stomach diseases, it would seem as though the results obtained could scarcely fail to bring out facts not shown by past experiments.

In the feeding experiments carried on in the McLean Hospital one such standard diet was adopted at the beginning and is still adhered to. The experience gained from a large number of cases has at various times suggested the desirability of introducing a few minor changes, as, for example, increasing somewhat the amount of salt contained in the diet and decreasing slightly the quantity of water given to women patients, but to do so would break the continuity of the experiment and it has therefore not been done.

The "laboratory diet" used is essentially a liquid food. The advantage of liquid as against the so-called solid food materials for exact feeding experiments is apparent. Such materials (milk and eggs) are not only far more constant in composition, necessitating therefore fewer analytical determinations, but they are the only kind that could be used in such a hospital as this, where many patients refuse all food and must be tube-fed, and where other patients can take only liquid food.

Each day's food is made up as follows:

500 cc. whole milk.
300 cc. cream (18-22% fat).
450 g. eggs (white and yolk).
20 g. sugar.
200 g. "Horlick's Malted Milk."
6 g. salt (75 cc. of a 10% solution).
Water enough to make two liters.

The above ingredients are all mixed together and make a fairly palatable mixture.

900 cc. of water is given in addition to what is contained in food. To the women patients is given the same quantity of water (900 cc.) but only two-thirds as much food.

known that corresponding differences in the amounts of sulphates or phosphates are obtained. The same may also be said with regard to certain products known to be rich in nucleins.

An explanation should perhaps be given for using a proprietary preparation like Horlick's Malted Milk. This preparation is, however, palatable and it was chosen because it was found to consist essentially of evaporated whey containing, therefore, little nitrogen and a great deal of milk sugar. As a carbohydrate food it seems to meet satisfactorily requirements difficult to fulfil, namely to furnish a palatable, non-nauseating, soluble carbohydrate.

From time to time samples of the food have been taken for analysis but it has not been considered necessary to do this very frequently because the nature of ingredients used and the method of preparation are such as to insure a constant diet for the conditions of these experiments.

If analyses of the feces had been included in the work it would of course also have been necessary to make daily analyses of the food. Such analyses, I regret to say, had to be left undone, because the large amount of additional work which this would involve could not be provided for.

Our records show the following analyses of the food:

Cl_2	SO_3	P_2O_5	N_2
6.25	3.69	5.75	18.92
6.02	3.71	5.54	18.80
....	3.62	5.92	18.86
....	3.81	5.92	18.86
....	3.80	....	19.05
....	3.84	....	19.05
....	....	....	19.02
....	....	....	19.02
....	....	....	19.02

General Description of the Feeding Experiments.[19]

The plan of the feeding experiments the analytical results of which are reported in this paper is as follows:

[19] Up to March, 1902, a large number of feeding experiments had been made with different diets. These experiments were intended as a study of the phosphate metabolism alone, because one of the first patients investigated showed unmistakably the presence of an abnormal phosphate metabolism. A brief report of this case was published in the American Journal of Physiology, 1902, vol. 7, p. 135, and will be referred to again in this paper.

A patient is selected by some member of the medical staff, though usually by Dr. A. Hoch, and more recently by Dr. F. Packard, who have also furnished the clinical descriptions accompanying the tables. A reliable special nurse (in difficult cases two nurses) is put in charge of the case, together with enough relief nurses to insure constant attention. At the beginning of the experiment and each subsequent morning the weight of the patient is taken. Two kilos of liquid food, prepared in laboratory, as well as a measuring cylinder (for the water) are given to the nurse in charge and in the course of the day (usually in three to five feedings) all the food and 900 cc. water are administered to the patient.

During the first two days that the patient is kept on the laboratory diet preliminary observations are made in regard to whether there is likely to be any difficulty in collecting all the urine, but the latter is not sent to the laboratory. On the morning of the third day, after voiding the bladder the collection of urine begins, each day ending at the same time the following morning. Since the collection of the urine does not begin till the third day of the experiment the urine may be said to fairly represent the food. It is, however, interesting to note that a number of the patients give on the third day a urine which, as judged by the several ratios and percentages, shows enough differences from the later urines to indicate that the two preliminary days were not quite enough to exhaust completely the influence of the food taken before the beginning of the experiment. No less than two and usually four or more 24-hour quantities of urine are then collected before stopping the experiment. In some cases it will be seen that the experiment has been continued for several weeks.[20]

Experiments with Normal Persons.

On the pages that follow will be found in tabular form the greater part of the analytical data obtained in the course of a large number of feeding experiments. In connection with a study of these tables attention must be called to the first eight. These

[20] In the more difficult cases the quantitative collection of the urine is accompanied by the use of a special cabinet chair devised many years ago by Dr. Cowles. The chair is supplied with a divided pan by means of which the urine and the feces are collected separately. This simple device has been especially useful in connection with the women patients.

represent seven fairly normal cases (six nurses and one of the doctors of the hospital). At the bottom of each table is given the averages of the different values obtained, and in Table 8 these averages are placed together and the final averages of all are given.

These normal cases, as well as many of the others, make exceedingly interesting material for study with a view of discovering the laws that govern the absolute and relative amount of each constituent excreted in the urine. For such a physiological study it is the variations even more than the correspondences that become important. It would be going too far out of the intended limits of this paper to give much discussion to this aspect of the analytical data here presented. It would seem appropriate to bring out one or two points, however, in order to illustrate the statements already made in this paper regarding the value of numerous complete urine analyses in connection with the use of uniform standard diets.

1. In the recent article of Krüger and Reich (Zeitschrift f. Physiol. Chemie, vol. 39, p. 179) on the determination of ammonia in urine is recorded a metabolism experiment lasting several days. The ammonia and the total nitrogen were determined and during four successive days the following percentages of the ammonia in terms of the total nitrogen were found: 6.18, 6.13, 6.22, 6.07. On the strength of these findings Krüger and Reich emphasize the importance of total nitrogen determination in connection with ammonia determinations because the uniformity of the the results indicate clearly, these authors think, that there must be a definite normal relationship between the amounts of total nitrogen and of ammonia eliminated through the urine. It is almost unnecessary to say that I agree with Krüger and Reich that ammonia determinations have more value when total nitrogen determinations also are made. The exact and definite relationship between these two constituents as found by Krüger and Reich is, however, as can be shown by these tables, accidental and is to be explained only on the ground of too few experiments.

More or less uniformity is certain to exist in the relation between almost any two urinary constituents under uniform conditions of diet and occupation, and unless this uniformity is far more persistent than is the relationship between total nitrogen and ammonia it proves nothing except the already known factors—constant diet and occupation. The fact that a uniform diet does of necessity tend to produce uniformity in nearly all ratios and percentages among the urinary constituents seems to have been overlooked in all or nearly all metabolism investigations and is responsible for many erroneous statements found in the literature on the subject.

That the ratio between total nitrogen and ammonia is not close enough to warrant the conclusion of Krüger and Reich can easily be seen from the accompanying tables. Variations in the percentages found from day to day are frequently from five to ten times as great as those reported by them and yet there is on the whole a pronounced tendency toward

uniformity. A stronger evidence, still, against the existence of such a relationship, is to be obtained by comparing different persons. Take, for example, the cases of Brown and Bogart (Tables 2 and 3). The former eliminated 18.24 g. of nitrogen per day with only 433 cc. tenth normal ammonia, while the latter eliminated only 16.6 g. of nitrogen and 608 cc. tenth normal ammonia per day. It is clear therefore that the laws governing the normal excretion of ammonia must be quite distinct from those governing the elimination of total nitrogen.

2. Table 7 representing the feeding experiment with Dr. P. illustrates another rather interesting point. From time to time Dr. P. had had attacks of articular rheumatism and in each case had obtained relief by taking sodium salicylate. At the time that this experiment was begun he had another attack but refrained from using the drug until the fifth day of the experiment, so as to enable us to see what effect it had on the uric acid elimination. The result is striking indeed. With the administration of 60 grains of sodium salicylate on the fifth day (January 29) the amount of uric acid eliminated was very nearly doubled. It is, however, further to be noted, first, that the elimination of uric acid was in this case fully up to the average of the other normal persons before taking the salicylate; and secondly, that the elimination of uric acid did not fall below the normal during the last two days of the experiment when no salicylate was taken. Both facts speak against Haig's view about the "storing up" of uric acid and rather indicate that sodium salicylate leads to an increased synthesis rather than to an increased elimination at the expense of uric acid already stored up in the system.

Simultaneous with the increase of uric acid in the urine on administering salicylate there is a very noticeable fall in the elimination of the chlorides. That this diminution in the elimination of the chlorides is not accidental is shown by the fact that it occurred again in one of the patients who received 50 grains of salicylate. I am unable to say whether or no this is a new observation or whether it has been made before. Contrary to expectation the indican is increased rather than diminished on the salicylate day. Another curious fact will be recorded here, as it will receive no further mention in this paper. In connection with the urea determinations we frequently find urines which turn blue on adding hydrochloric acid (without the addition of any oxidizing reagent whatever). It is clear that in such urines the indican must be present in a form quite different from what is ordinarily the case. In some patients hot baths have so changed the urines of the patients that strong indican reactions were obtained with hydrochloric acid alone. The sodium salicylate produced a similar change in the urine of Dr. P. A strong indican reaction was obtained with HCl alone on January 29 and the reaction was negative on all the other days of the experiment.

It is evident from the analytical data of this table that sodium salicylate must have a very pronounced influence on the metabolism.

No. 1. Name, MR. MAX HUWYLER. (Normal.)

Weight at the beginning of the experiment, 56.600 k.
" " end " " 56.500 k.
Loss ..... 100 g.

1903. 24 hours ending	Sp. gr. 1.0—	P_2O_5, mg. in 50 cc.	SO_3, mg. in 50 cc.	Cl_2, mg. in 10 cc.	Ethereal SO_3, mg. in 50 cc.	Neutral SO_3, mg. in 50 cc.	N_2, mg. in 5 cc.	Urea, grms.	NH_3, cc. $\frac{N}{10}$	Kreatinin, grms.	Uric acid, gm.	Acidity cc. $\frac{N}{10}$ Mineral.	Organic.	Indican.	Ratios. 100 N_2 : P_2O_5	100 N_2 : SO_3	100 N_2 : Cl_2	100 SO_3 : P_2O_5	100 SO_3 : Ethereal SO_3	100 SO_3 : Neutral SO_3	Per cent of total N_2 Urea.	NH_3	Kreatinin.	Uric acid.	Rest.
August	cc. in 24°	Total	Total	Total	Total	Total	Total	N. Total	N. Total	N. Total	N. Total	Titrated. (Total)										Sum				
23	17.5 970	162.5 3.15	14.7 2.86	62 6.01	0.5 .203	2.3 .045	61.7 11.97	21.98 10.26	330 .46	1.42 .53	.430 .143	262 563	301	25	26.3	23.9	50.2	110.2	7.1	1.5	85.51 89.34	3.83 3.4	4.4	1.2	5.06
24	18 1040	142 2.95	1401 2.90	66 6.86	7.9 .163	4 .082	69.3 14.41	27.43 12.8	362 .51	1.31 .50	.478 .159	120 499	379	25	20.47	20.1	47.6	102	5.6	2.8	89.3 92.73	3.4 4	3.5	1.1	2.7
25	20 1590	119 3.78	100 3.18	45.4 7.12	7.1 .225	4.2 .134	51.2 16.35	31.50 14.7	470 .66	1.38 .51	.518 .173	311 686	375	20	22.43	18.9	43.8	119	7.1	4.2	90 94	4	3.15	1.05	2
26	14.5 2000	81.5 3.26	67.6 2.70	32.7 6.53	4.8 .192	3.2 .128	34.7 13.88	26.49 12.36	352 .49	1.22 .45	.348 .116	316 552	236	3	23.27	19.45	47.0	120.8	7	4.7	89.11 92.74	3.63	3.25	.85	3.15
27	19 1665	112 3.73	100. 0 3.35	42.6 7.09	5.9 .196	3.4 .113	45.8 15.15	28.93 13.5	392 .55	1.38 .51	.470 .157	226 613	387	10	24.6	22.1	46.8	111	5.8	3.3	89.37 92.97	3.60	3.4	1.04	3
28	17.5 1930	97.5 3.76	86 3.31	36.1 6.97	4.3 .166	8.8 .339	41. 15.82	30.22 14.1	386 .53	1.5 .56	.500 .167	255 672	417	3	23.76	20.9	44.1	113	5	10.2	89.11 92.52	3.41	3.5	1.06	2.9
Average of last 5 days.	**1645**	**3.44**	**3.08**	**6.9**	**.188**	**.159**	**15.1**	**28.93** **13.5**	**392** **.55**	**1.36** **.504**	**.462** **.154**	**247** **604**	**359**	**12**	**22**	**20.4**	**46.6**	**112**	**6**	**5**	**89.4** **93**	**3.6**	**3.33**	**1**	**2.7**

No. 2. Name, MR. S. R. BROWN. { Weight, September 13.....57.784 k.
" " 18.....58.170 k.
Gain..... 386 g.

1903. 24 hours ending	Sp. gr. 1.0—	P_2O_5, mg. in 50 cc.	SO_3, mg. in 50 cc.	Cl_2, mg. in 10 cc.	Ethereal SO_3, mg. in 50 cc.	Neutral SO_3, mg. in 50 cc.	N_2, mg. in 5 cc.	Urea, gms.	NH_3, cc. $\frac{N}{10}$	Kreatinin, gms.	Uric acid, gm.	Acidity cc. $\frac{N}{10}$ Mineral.	Organic.	Indican.	Ratios. 100 N_2 : P_2O_5	100 N_2 : SO_3	100 N_2 : Cl_2	100 SO_3 : P_2O_5	100 SO_3 : Ethereal SO_3	100 SO_3 : Neutral SO_3	Per cent of total N_2 Urea	NH_3	Kreatinin.	Uric acid.	Rest.
Sept.	cc. in 24°	Total	Total	Total	Total	Total	Total	N. Total	N. Total	N. Total	N. Total	Titrated. (Total)									Sum				
13	28.5 1065	191.5 4.07	162.3 3.56	49.7 5.30	8.3 .177	10 .210	78.1 16.6	31.25 14.54	428 .60	1.53 .57	.44 .140	264 618	354	95	24.5	21.4	31.9	114	5	5.9	87.6 91.7	3.6	3.4	.9	4.5
14	27.5 1230	177.5 4.37	144.7 356	49.3 6.06	10.7 .262	7.4 .180	79.2 19.5	36.86 17.2	443 .62	1.6 .595	.45 .15	310 674	364	100	22.4	18.8	31.1	119	7.1	5.1	88.3 91.5	3.2	3.1—	.8	4.6
15	25.5 1290	170 4.39	133.5 3.44	51.1 6.59	11.1 .288	7.2 .190	69.6 18	34.71 16.2	345 .48	1.55 .576	.48 .16	150 542	392	110	24.3	19.1	36.6	127	8.4	5.5	90.1 92.8	2.7	3.2	.9	3.
16	28 1240	206 5.01	159 3.94	44.0 5.46	15.4 .39	....	78.1 19.4	37.07 17.3	511 .715	1.64 .61	.46 .15	367 768	401	110	25.8	20.3	28.1	135	9.9	....	89.3 92.8	3.5	3.15	.8	.25
17	27 1370	170 4.66	132.1 3.52	51.1 7.0	12.1 .33	8.7 .28	64.5 17.7	33.85 15.8	438 .61	1.6 .595	.40 .13	 652	....	110	26.3	20	39.5	132	9.4	7.9	89.4 92.8	3.4	3.34	.75	2.9
Average of last 5 days.	1239	4.5	3.65	6.08	.29	.215	18.24	34.71 16.2	433 .603	1.58 .59	.438 .146	273 651	378	105	24.6	20	33.4	123	8	6.1	88.84 92.16	3.32	3.23	.83	3.66

No. 3. Name, MR. JOHN H. BOGART. { Weight, September 8.....57.436 k. " " 12.....57.436 k. Gain..... 0

1903. 24 hours ending	Sp. gr. 1.0—	P_2O_5, mg. in 50 cc.	SO_3, mg. in 50 cc.	Cl_2, mg. in 10 cc.	Ethereal SO_3, mg. in 50 cc.	Neutral SO_3, mg. in 50 cc.	N_2, mg. in 5 cc.	Urea, grms.	NH_3, cc. $\frac{N}{10}$	Kreatinin, grms.	Uric acid, grm.	Acidity cc. $\frac{N}{10}$ Mineral.	Acidity cc. $\frac{N}{10}$ Organic.	Indican.	Ratios. 100 N_2 : P_2O_5	Ratios. 100 N_2 : SO_3	Ratios. 100 N_2 : Cl_2	Ratios. 100 SO_3 : P_2O_5	Ratios. 100 SO_3 : Ethereal SO_3	Ratios. 100 SO_3 : Neutral SO_3	Per cent of total N_2 Urea.	Per cent of total N_2 NH_3	Per cent of total N_2 Kreatinin.	Per cent of total N_2 Uric acid.	Per cent of total N_2 Rest.
Sept.	cc. in 24°	Total	Total	Total	Total	Total	Total	N. Total	N. Total	N. Total	N. Total	Titrated. (Total)										Sum				
7	17.5 1980	91.5 3.62	81.2 3.22	36.9 7.30	4.5 .178	4.6 .182	42.4 16.8	28.93 13.5	649 .91	1.54 .57	.332 .111	... 515	...	10	21.6	19.1	43.4	112	5.5	5.7	86.3 91.7	5.4	3.4	.7	4.2
8	19 1840	101.5 3.73	92.1 3.39	35.5 6.53	5.5 .202	4.7 .173	45.9 16.9	31.72 14.8	684 .96	1.47 .55	.294 .098	140 574	434	70	22.1	20	39.3	110	5.9	4.7	87.6 93.3	5.7	3.2	.6	2.9
9	20 1655	108 3.57	98.1 3.25	36.9 6.10	5.6 1.87	4.1 .152	51.2 17.19	32.51 15.17	583 .815	1.6 .6	.364 .12	298 550	252	85	20.8	19	35.7	110	5.2	4.2	88.2 93	4.8	3.9	.7	2.4
10	17 1770	107 3.79	90.2 3.19	32 5.67	5.4 1.93	6.4 .23	46.3 16.3	30.62 14.29	580 .812	1.56 .58	.343 .114	205 580	375	80	23.3	19.6	34.7	119	5.1	7.2	87.7 92.7	5	3.6	.7	3
11	16 1815	103.5 3.76	87.5 3.18	32 5.81	4.9 .18	2.5 .09	43.7 15.9	29.79 13.9	546 .764	1.7 .63	.337 .112	174 552	378	12	23.6	20	36.5	118	5.7	2.9	87.5 92.3	4.8	4	.7	3
Average of last 5 days.	1812	3.69	3.25	6.38	.188	.165	16.6	30.64 14.3	608 .85	1.57 .59	.33 .11	204 554	360	51	22.3	19.5	37.9	114	5.5	4.9	87.5 92.6	5.1	3.6	.68	3.1

No. 4. Name, Mr. E. H. Scott. { Weight, September 29.....67.057 k.
" October 4.....66.149 k.
Loss..... 908 g.

1903. 24 hours ending	Sp. gr. 1.0—	P_2O_5, mg. in 50 cc.	SO_3, mg. in 50 cc.	Cl_2, mg. in 10 cc.	Ethereal SO_3, mg. in 50 cc.	Neutral SO_3, mg. in 50 cc.	N_2, mg. in 5 cc.	Urea, grms.	NH_3, cc. $\frac{N}{10}$	Kreatinin, grms.	Uric acid, gm.	Acidity cc. $\frac{N}{10}$ Mineral.	Organic.	Indican.	Ratios. 100 N_2 : P_2O_5	100 N_2 : SO_3	100 N_2 : Cl_2	100 SO_3 : P_2O_5	100 SO_3 : Ethereal SO_3	100 SO_3 : Neutral SO_3	Per cent of total N_2 Urea.	NH_3	Kreatinin.	Uric acid.	Rest.
Sept.	cc. in 24°	Total	Total	Total	Total	Total	Total	N. Total	N. Total	N. Total	N. Total	Titrated. (Total)										Sum				
29	27 1120	161 3.61	128 2 87	60.4 6.76	10.3 .231	9.5 .21	62 13.9	25.89 12.08	493 .690	1.41 .530	.189 .63	398	207 605	90	24.2	20.6	48.5	125.8	8	7.4	87	4.9 91.87	3.8	.45	3.8
30	18.5 1740	110 3.86	82.4 2.87	37.6 6.54	7 .245	6.2 .22	44.2 15.38	28.65 13.37	543 .760	1.5 .56	.258 .86	403	265 668	90	25.2	18.6	42.5	135	8.4	7.7	87.1	4.8 91.9	3.6	.56	4.9
Oct. 1	22.5 1260	148.5 3.74	114 2.88	41.9 5.28	9.4 .236	8.1 .20	57.7 14.54	26.72 12.47	486 .680	1.49 .55	.198 .66	378	302 680	90	25.7	19.8	36.2	130	8.2	6.9	85.8	4.7 90.5	3.8	.45	5.2
2	29.5 900	213.5 3.84	162.4 2.92	56.8 5.11	14.8 .266	9.6 .173	79.2 14.26	25.82 12.05	475 .665	1.44 .54	.223 .74	435	265 698	80	26.9	20.4	35.7	132	9.1	5.9	84.5	4.7 89.2	3.8	.55	6.4
3	32 960	211.5 4.06	162.4 3,12	61.1 5.86	15 .288	5.2 .10	83.2 15.87	29.72 13.87	465 .651	1.42 .53	.330 .11	472	223 695	80	25.6	19.7	31.5	130	9.2	3.2	87.3	4.1 91.4	3.3—	.69	4.6
Average of last 5 days.	**25.9** **1196**	**3.82**	**2.93**	**5.91**	**.253**	**.19**	**14.8**	**27.34** **12.76**	**492** **.690**	**1.46** **.54**	**.24** **.08**	**417**	**252** **669**	**86**	**25.5**	**19.8**	**39.9**	**131**	**8.6**	**6.2**	**86.3**	**4.7** **91**	**3.66**	**.54**	**5.0**

No. 5. Name, MR. R. L. JONES. { Weight, September 21..... 70.824 k. " " 26..... 70 143 k. Loss..... 681 g.

1903. 24 hours ending	Sp. gr. 1.0—	P_2O_5, mg. in 50 cc.	SO_3, mg. in 50 cc.	Cl_2, mg. in 10 cc.	Ethereal SO_3, mg. in 50 cc.	Neutral SO_3, mg. in 50 cc.	N_2, mg. in 5 cc.	Urea, grms.	NH_3, cc. $\frac{N}{10}$	Kreatinin, grms.	Uric acid, gm.	Acidity cc. $\frac{N}{10}$ Mineral.	Acidity cc. $\frac{N}{10}$ Organic.	Indican.	Ratios. 100 N_2 : P_2O_5	Ratios. 100 N_2 : SO_3	Ratios. 100 N_2 : Cl_2	Ratios. 100 SO_3 : P_2O_5	Ratios. 100 SO_3 : Ethereal SO_3	Ratios. 100 SO_3 : Neutral SO_3	Per cent of total N_2 Urea.	Per cent of total N_2 NH_3.	Per cent of total N_2 Kreatinin.	Per cent of total N_2 Uric acid.	Per cent of total N_2 Rest.
Sept.	cc. in 24°	Total	Total	Total	Total	Total	Total	N. Total	N. Total	N. Total	N. Total	Titrated. (Total)										Sum				
21	195	131	102	41.6	8.1	7	52.2	29.27	466	1.64	.236	219	370	140	25	19.5	39.7	128	7.9	6.9	85.9	4.1	3.8	.47	5.7
	1520	3.98	3.10	6.32	.245	.21	15.9	13.66	.644	.61	.079	589									90				
22	21	136	106	36.9	8.3	.....	54.3	31.08	514	1.56	.304	299	331	150	25.1	19.6	34.0	128	7.7	....	86.9	4.3	3.6	.61	5.4
	1530	4.16	3.25	5.65	.252	.....	16.6	14.48	.720	.58	.101	630									91.2				
23	22	131.5	108.7	39.7	9.5	9	56.4	30.43	520	1.53	.328	432	193	140	23.2	19.2	35.2	128	8.8	5.7	86.5	4.4	3.4	.66	5.0
	1460	3.84	3.17	5.80	.279	.18	16.6	14.20	.728	.56	.109	625									90.9				
24	24	138.5	119.4	42.6	8.9	4.7	61.9	30.39	532	1.67	.375	....	....	140	22.3	19.3	34.4	116	7.4	4.2	86.1	4.5	3.2	.76	5.5
	1330	3.68	3.07	5.67	.237	.13	16.5	14.18	.745	.52	.125	617									90.6				
25	23	139.5	113.5	37.6	7.8	4.3	60.2	30.51	618	1.45	.328	276	370	130	23.2	19	31.2	123	6.8	3.9	85.7	5.2	3.3	.66	5.1
	1380	3.85	3.13	5.19	.214	.12	16.6	14.24	.865	.54	.109	646									90.9				
Average of last 5 days.	**1444**	**3.90**	**3.15**	**5.72**	**.245**	**.16**	**16.4**	**30.32** 14.15	**529** .741	**1.57** .59	**.314** .105	**308**	**313**	**140**	**23.7**	**19.3**	**34.9**	**123**	**7.7**	**5.2**	**86.2**	**4.5**	**3.5**	**.63**	**5.3**
												621									**90.7**				

No. 6. Name, MR. GEO. E. CAMPBELL. { Weight, August 31.....70.940 k. " Sept. 5.....71.143 k. Gain.....203 g.

1903. 24 hours ending	Sp. gr. 1.0—	P_2O_5, mg. in 50 cc.	SO_3, mg. in 50 cc.	Cl_2, mg. in 10 cc.	Ethereal SO_3, mg. in 50 cc.	Neutral SO_3, mg. in 50 cc.	N_2, mg. in 5 cc.	Urea, gms.	NH_3, cc. $\frac{N}{10}$	Kreatinin, grms.	Uric acid, gm.	Acidity cc. $\frac{N}{10}$ Mineral.	Acidity cc. $\frac{N}{10}$ Organic.	Indican.	Ratios. 100 N_2 : P_2O_5	Ratios. 100 N_2 : SO_3	Ratios. 100 N_2 : Cl_2	Ratios. 100 SO_3 : P_2O_5	Ratios. 100 SO_3 : Ethereal SO_3	Ratios. 100 SO_3 : Neutral SO_3	Per cent of total N_2 Urea.	Per cent of total N_2 NH_3.	Per cent of total N_2 Kreatinin.	Per cent of total N_2 Uric acid.	Per cent of total N_2 Rest.
Aug.	cc. in 24°	Total	Total	Total	Total	Total	Total	N. Total	N. Total	N. Total	N. Total	Titrated. (Total)											Sum			
31	18.5	117.5	89.4	39.1	5.7	3.2	46.6	30.58	571	1.88	.417	257	362	14	25.2	19.2	41.9	131.5	7.7	4.6	87.9	4.9	4.3	.86	2
	1740	4.09	3.11	6.8	.24	.11	16.22	14.27	.80	.7	.14		619									92.8			
Sept. 1	24	168	122	47.6	8.8	4.8	61.7	27.92	542	1.74	.413	410	287	85	27.2	19.8	38	137.6	7.2	4	86.6	5	4.3	.92	3.2
	1220	4.10	2.98	5.8	215	.117	15.05	13.03	.76	.65	.14		697									91.6			
2	26	191	142.7	51.8	8	.....	68.6	25.78	485	1.67	.360	412	226	85	27.8	20.5	37.5	137	5.8	.....	86.7	4.9	4.5—	.86	3
	1010	3.86	2.84	5.2	.166	.....	13.86	12.03	.680	.62	.12		638									91.6			
3	20	148	107.5	41.2	6.4	4.9	55.7	27.58	606	1.72	.359	388	260	80	26.6	19.3	36.9	138	6	4.9	87	4.7	4.3	.8	3.2
	1330	3.94	2.86	5.47	.17	.140	14.8	12.87	.848	.64	.12		648									91.7			
4	28.5	210	163.6	50.4	9.9	7.4	77.1	25.72	539	.182	.624	408	283	87	27.2	21.2	32.8	129	6	4.4	85.4	5.4	4.9—	1.48	2.7
	910	3.82	2.98	4.59	.18	.130	14.04	12	.75	.68	.208		691									90.9			
Average of last 5 days	1242	3.96	2.95	5.6	.194	.125	14.9	27.42 12.84	549 .77	1.77 .66	.438 .146	375	284 601	70	26.6	20	37.2	134.6	6.5	4.5	86.7	5.0	4.5	.98	2.8
																						91.7			

No. 5. Name, MR. R. L. JONES. { Weight, September 21..... 70.824 k. " " 26..... 70 143 k. Loss..... 681 g.

1903. 24 hours ending	Sp. gr. 1.0—	P_2O_5, mg. in 50 cc.	SO_3, mg. in 50 cc.	Cl_2, mg. in 10 cc.	Ethereal SO_3, mg. in 50 cc.	Neutral SO_3, mg. in 50 cc.	N_2, mg. in 5 cc.	Urea, grms.	NH_3, cc. $\frac{N}{10}$	Kreatinin, grms.	Uric acid, gm.	Acidity cc. $\frac{N}{10}$ Mineral.	Acidity cc. $\frac{N}{10}$ Organic.	Indican.	Ratios. 100 N_2 : P_2O_5	100 N_2 : SO_3	100 N_2 : Cl_2	100 SO_3 : P_2O_5	100 SO_3 : Ethereal SO_3	100 SO_3 : Neutral SO_3	Per cent of total N_2 Urea.	Per cent of total N_2 NH_3	Kreatinin.	Uric acid.	Rest.
Sept.	cc. in 24°	Total	Total	Total	Total	Total	Total	N. Total	N. Total	N. Total	N. Total	Titrated. (Total)										Sum				
21	195 1520	131 3.98	102 3.10	41.6 6.32	8.1 .245	7 .21	52.2 15.9	29.27 13.66	466 .644	1.64 .61	.236 .079	219	370 589	140	25	19.5	39.7	128	7.9	6.9	85.9 90	4.1	3.8	.47	5.7
22	21 1530	136 4.16	106 3.25	36.9 5.65	8.3 .252	....	54.3 16.6	31.03 14.48	514 .720	1.56 .58	.304 .101	299	331 630	150	25.1	19.6	34.0	128	7.7	....	86.9 91.2	4.3	3.6	.61	5.4
23	22 1460	131.5 3.84	108.7 3.17	39.7 5.80	9.5 .279	9 .18	56.4 16.6	30.43 14.20	520 .728	1.53 .56	.328 .109	432	193 625	140	23.2	19.2	35.2	128	8.8	5.7	86.5 90.9	4.4	3.4	.66	5.0
24	24 1330	138.5 3.68	119.4 3.07	42.6 5.67	8.9 .237	4.7 .13	61.9 16.5	30.39 14.18	532 .745	1.67 .52	.375 .125	....	 617	140	22.3	19.3	34.4	116	7.4	4.2	86.1 90.6	4.5	3.2	.76	5.5
25	23 1380	139.5 3.85	113.5 3.13	37.6 5.19	7.8 .214	4.3 .12	60.2 16.6	30.51 14.24	618 .865	1.45 .54	.328 .109	276	370 646	130	23.2	19	31.2	123	6.8	3.9	85.7 90.9	5.2	3.3	.66	5.1
Average of last 5 days.	**1444**	**3.90**	**3.15**	**5.72**	**.245**	**.16**	**16.4**	**30.32** **14.15**	**529** **.741**	**1.57** **.59**	**.314** **.105**	**308**	**313** **621**	**140**	**23.7**	**19.3**	**34.9**	**123**	**7.7**	**5.2**	**86.2** **90.7**	**4.5**	**3.5**	**.63**	**5.3**

No. 6. Name, MR. GEO. E. CAMPBELL. { Weight, August 31.....70.940 k. " Sept. 5.....71.143 k. Gain.....203 g.

1903. 24 hours ending	Sp. gr. 1.0—	P_2O_5, mg. in 50 cc.	SO_3, mg. in 50 cc.	Cl_2, mg. in 10 cc.	Ethereal SO_3, mg. in 50 cc.	Neutral SO_3, mg. in 50 cc.	N_2, mg. in 5 cc.	Urea, gms.	NH_3, cc. $\frac{N}{10}$	Kreatinin, gms.	Uric acid, gm.	Acidity cc. $\frac{N}{10}$ Mineral.	Organic.	Indican.	Ratios. 100 N_2 : P_2O_5	SO_3	Cl_2	100 SO_3 : P_2O_5	Ethereal SO_3	Neutral SO_3	Per cent of total N_2 Urea.	NH_3	Kreatinin.	Uric acid.	Rest.
Aug.	cc. in 24°	Total	Total	Total	Total	Total	Total	N. Total	N. Total	N. Total	N. Total	Titrated. (Total)									Sum				
31	18.5 1740	117.5 4.09	80.4 3.11	39.1 6.8	5.7 .24	3.2 .11	46.6 16.22	30.58 14.27	571 .80	1.88 .7	.417 .14	257 619	362	14	25.2	19.2	41.9	131.5	7.7	4.6	87.9 4.9 92.8		4.3	.86	2
Sept. 1	24 1220	168 4.10	122 2.98	47.6 5.8	8.8 215	4.8 .117	61.7 15.05	27.92 13.03	542 .76	1.74 .65	.413 .14	410 697	287	85	27.2	19.8	38	137.6	7.2	4	86.6 5 91.6		4.3	.92	3.2
2	26 1010	191 3.86	142.7 2.84	51.8 5.2	8 .166	....	68.6 13.86	25.78 12.03	485 .680	1.67 .62	.360 .12	412 638	226	85	27.8	20.5	37.5	137	5.8	.....	86.7 4.9 91.6		4.5—	.86	3
3	20 1330	148 3.94	107.5 2.86	41.2 5.47	6.4 .17	4.9 .140	55.7 14.8	27.58 12.87	606 .848	1.72 .64	.359 .12	388 648	260	80	26.6	19.3	36.9	138	6	4.9	87 4.7 91.7		4.3	.8	3.2
4	28.5 910	210 3.82	163.6 2.98	50.4 4.59	9.9 .18	7.4 .130	77.1 14.04	25.72 12	539 .75	.182 .68	.624 .208	408 691	283	87	27.2	21.2	32.8	129	6	4.4	85.4 5.4 90.9		4.9—	1.48	2.7
Average of last 5 days.	1242	3.96	2.95	5.6	.194	.125	14.9	27.42 12.84	549 .77	1.77 .66	.438 .146	375 601	284	70	26.6	20	37.2	134.6	6.5	4.5	86.7 5.0 91.7		4.5	.98	2.8

No. 7. Dr. P. { Weight, January 27 ..... 54.720 k.
" February 1..... 55.692 k.
Gain..... 972 g.

1903. 24 hours ending	Sp. gr. 1.0—	P_2O_5, mg. in 50 cc.	SO_3, mg. in 50 cc.	Cl_2, mg. in 10 cc.	Ethereal SO_3, mg. in 50 cc.	Neutral SO_3, mg. in 50 cc.	N_2, mg. in 5 cc.	Urea, gms.	NH_3, cc. $\frac{N}{10}$	Kreatinin, gms.	Uric acid, gm.	Acidity cc. $\frac{N}{10}$ Mineral.	Organic.	Indican.	Ratios. 100 N_2 : P_2O_5	100 N_2 : SO_3	100 N_2 : Cl_2	100 SO_3 : P_2O_5	100 SO_3 : Ethereal SO_3	100 SO_3 : Neutral SO_3	Per cent of total N_2 Urea.	NH_3	Kreatinin.	Uric acid.	Rest.
Jan.	cc. in 24°	Total	Total	Total	Total	Total	Total	N. Total	N. Total	N. Total	N. Total	Titrated. (Total)									Sum				
27	16 1545	111 3.43	88 2.72	39.5 6.14	.....	.....	46 14.22	26.65 12.44	365 .511	.....	.376 .125	.....	.....	—	24.1	19.1	43.2	126	.....	.....	87.5	3.6	.....	.9	.....
												633		33											
28	16 1600	130 4.16	99 3.17	35.5 5.68	.....	.....	51 16.22	30.06 14.03	384 .538	.....	446 .149	.....	.....	—	25.6	19.5	35	131	.....	.....	91.1 86.5	3.4	.....	.9	.....
												720		33											
29*	23 1275	143 3.64	127 3.24	29 3.70	.....	.....	66 16.88	31.86 14.87	357 .500	.....	850 .283	.....	.....	+	21.6	19.2	21.9	112	.....	.....	89.8 88.07	3	.....	1.7	.....
												586		60											
30	25 1100	170 3.74	133 2.93	49.5 5.44	.....	.....	75 16.54	31 14.47	418 .585	.....	.385 .128	.....	.....	—	22.6	17.7	33.6	127	.....	.....	91.07 88.14	3.56	.....	.8	.....
												704		25											
31	18 1450	131 3.83	107 3.10	44.5 6.48	.....	.....	55 15.83	29.46 13.70	487 .681	.....	.322 .107	.....	.....	—	24.2	19.6	40.9	124	.....	.....	91.7 86.5	4.3	.....	.7	.....
												678		60							90.8				
Average of last 5 days.	1394	3.76	3.03	5.49	.....	.....	15.94	29.78 13.90	402 .563	.....	.382 .127	.....	.....	42	23.6	19	34.9	124	.....	.....	87.36	3.54	.....	.82	.....
												664									90.9				

* Sodium salicylate administered.

In looking over the tables representing the feeding experiments with the normal persons it will be seen that, while there is a tendency toward uniformity in nearly all the absolute values as well as in the different ratios and percentages, there are also variations which must be taken into account and considered normal. In connection with the tables obtained from similar feeding experiments with the patients, it will be found that the corresponding values and relationships vary within very much wider limits than those set by the seven normal persons.

$\frac{N_2}{SO_3}$ Among the different relationships recorded, the ratio between the total nitrogen and the ordinary sulphates is remarkably constant for the normal persons and, indeed, also for most of the patients (the highest normal ratio being 100 : 20.4, the lowest 100 : 19.1). In view of this constancy, which one would naturally expect from a constant diet, it is certainly interesting and instructive to find that even this ratio is, in some of the patients, represented by figures far outside the variations found among the normal persons. This subject will be referred to again in connection with the abnormal cases.

$\frac{N_2}{\text{Urea} + \text{Ammonia}}$ Another ratio even more constant than that of the nitrogen-sulphate ratio is the percentage of the urea-ammonia nitrogen in terms of the total nitrogen (the highest normal being 93, the lowest 91). As far as I am aware, there is no record in the literature of the fact that the sum of the nitrogen belonging to the urea and the ammonia is far more constant, as a percentage of the total nitrogen, than is the nitrogen of either constituent taken by itself. It would be impracticable to include a full discussion of this interesting fact in the present paper and I therefore confine myself to calling the reader's attention to it. The ratio is almost as constant for the patients as it is for the normal cases, notwithstanding the fact that very pronounced variations occur in the percentages of urea and ammonia taken singly.

$\frac{N_2}{\text{Kreatinin and Uric Acid}}$ The ratios of kreatinin nitrogen, and, to a less degree, of the uric acid nitrogen to the total nitrogen, vary also within relatively narrow limits in the normal persons

and again appear as decidedly fluctuating factors in the metabolism records of the patients.

$\frac{N_2}{\text{Undetermined } N_2}$ Particular attention must be called to the last column in the tables representing the undetermined residue of nitrogenous substances. In the planning of these researches it was hoped that a systematic scheme of analysis, including the determination of all the more important nitrogenous constituents and accounting, as we find it does, for about 96 per cent of the nitrogen in terms of known products, would leave an undetermined nitrogenous residue so small that the occurrence in it of any notable quantity of some abnormal product would be detected. If any such substance should occur and should be an organic base, such as several investigators (notably Griffith) state that they have found in the urines of the insane, this fact it was thought would be indicated on the one hand by a high percentage of undetermined nitrogen and on the other by a high mineral acidity as compared with the total acidity and the organic acidity.[n]

From a study of the records of the patients it will be seen that the undetermined nitrogenous residue is in several cases somewhat greater than the normal, thus indicating that among these undetermined substances must be one or more formed wholly or in part at the expense of the more important known nitrogen compounds, usually urea or kreatinin. This will be referred to again in connection with the records of the patients.

$\frac{N_2}{Cl_2}$ It may seem rather strange that the ratio of total nitrogen to total chlorine should be included in these records, since these two constituents may be supposed to have but little in com-

[n] If such an organic base should be accompanied by an increase of organic acids the acidity determinations would of course fail to indicate its presence and only the nitrogen determinations could be expected to give the sought-for indication. Whether or no this would be likely to be the case it is impossible to say. The fact that organic acids are as much, or more, important than the mineral acids in the make-up of the total acidity of normal urine was not known when these investigations were begun. This fact was discovered in connection with the mineral acidity determinations. (See the acidity paper, loc. cit., p. 278.)

mon and to be excreted by the kidney, according to entirely independent laws. Forster's hypothesis, according to which a certain part of the chlorides are held in combination with the proteins of the serum, a hypothesis accepted by Sollman[22] might, however be cited in justification of the ratio. At any rate the relationship illustrates what has already been said in regard to the tendency of almost all urinary ratios to become comparatively uniform with a uniform diet. It will be noted that in the normal cases the chloride ratio is fairly constant, notwithstanding the fact that the volumes of urine excreted by the different persons differ within very wide limits.

Together with the chloride-nitrogen ratio should perhaps have been introduced another, giving the relationship of the chlorides to the volume of the urine since it is generally supposed that the amount of chlorine excreted depends to a very great extent upon the amount of water eliminated. The same relationship is, however, fairly represented in the tables by the amount of chlorine found in 10 cc. of urine and this amount is not very constant.

$\frac{N_2}{P_2O_5}$ and $\frac{SO_3}{P_2O_5}$ No relationship among the urinary constituents has been studied so persistently as that of the phosphates to the total nitrogen and the total sulphates. This subject must be treated rather more fully at the end of this paper, and it is, therefore, enough to refer here to the fact that the variations in these two ratios are far greater among the patients than among the normal cases recorded above.

VOLUME OF URINE.—Though it is not my intention to emphasize in this paper the normal physiological aspects of the various data recorded in these tables, I cannot refrain from pointing out one striking fact which has especially engaged my attention. I refer to the volume of urine excreted. In the last number of the American Journal of Physiology (Nov., 1903) Hawk reports findings corresponding to those to be mentioned here, and devotes some discussion to the subject. It is generally assumed, as Hawk correctly remarks, that the volume of urine excreted during any given 24-hour period is more or less exactly proportional to the

[22] Am. Journ. of Physiology, vol. 9, p. 448 (1903).

amount of liquid ingested under normal or uniform conditions of occupation and environment. Nothing could, however, be further from the fact. No other urinary constituent shows such capricious, such unaccountable variations from day to day and such differences when different persons are compared. (See for examples Table 2 and 3). Numerous careful observations on the atmospheric conditions with regard to temperature and moisture, as well as with regard to daily gain or loss of body weight, have convinced me that none of these conditions exerts more than a relatively small influence on the volume of urine excreted.

The cause of this remarkable variation in the amount of urine eliminated must be sought in corresponding variations in the quantities of water eliminated through the lungs or the skin. Some investigations on this subject, with reference to the lungs, were made during the summer of 1902, and considerable variations were found, but the conditions of those experiments were not such as to insure free and natural respiration and no conclusions could be drawn from the results. This question is, I venture to say, very important in connection with the metabolism of the insane, and I hope to be able to take it up in connection with the feeding experiments. It seems to me that the remarkable gains and losses of weight which are observed in this kind of patients may, to a very large extent, be due to diminished or increased elimination of water through the lungs or through the skin.

(To be continued.)

THE CHEMICAL FINDINGS IN THE CEREBRO-SPINAL FLUID AND CENTRAL NERVOUS SYSTEM IN VARIOUS MENTAL DISEASES.

By ISADOR H. CORIAT, M. D.,
Worcester Insane Hospital.

I. Introduction.

In a previous communication upon this subject (1), I had shown that cholin, one of the katabolic derivatives of the lecithin, which forms the principal substance of the myelin sheaths of nerves, was present in the cerebro-spinal fluid, blood, and central nervous system in those pathological conditions where active degeneration had taken place. This was independent of the lapse of time between the analysis and the duration of the disease process, being found alike in general paralysis, and the organic dementias of several years' duration, as well as in those various depressive disorders with a termination in central neuritis, where the degeneration was both recent and active. At the same time, where the nature of the disease or the amounts of the cerebro-spinal fluid seemed to warrant it, the analysis was carried further, and the specific gravity, the reducing body, the presence or the absence of lactic acid, and an approximate conclusion as to the amount of proteid present, were noted. The presence of cholin and the chemical basis of the Marchi reaction, are dependent upon the splitting-up of the lecithin. These findings, which I have briefly cited, were, however, interpreted from a qualitative standpoint, in conjunction with whatever physical or chemical characteristics of the cerebro-spinal fluid that would be conducive to its elucidation. Since this paper has been published, however, I have been able to carry my researches further along this line, making the analysis both qualitative and quantitative, investigating special problems, which inadvertently crop up in a work of this nature, and furthermore have added a study of the cerebro-

spinal fluid from the standpoint of physical chemistry. This has been partly the result of stimulation from the growing literature on the subject and partly of the inevitable evolution which takes place along certain lines, when special problems are investigated. In this paper it is proposed to deal in detail with what had been previously merely touched upon, and to give a further elaboration, and I hope elucidation, of the increasingly important subject of nerve chemistry. It is only after a weighing and rigid comparison of chemical, pathological and clinical factors, that definite conclusions can be drawn.

The renewed interest in nerve chemistry during recent years, and the efforts to marshal in order the former confusion of facts, makes the problem a wide one. Studies of tracts and cells are of great importance, but they have contributed nothing to the fundamental causes of various nervous and mental phenomena. Neuro-chemistry can, however, be of incalculable assistance to neuro-pathology by laying bare the intimate and intricate chemical structures of the most complex of tissues, and thus establish a new path to explain the chemical and metabolic basis of disease, as recent investigations have abundantly illustrated. I refer to the work of Koch in his admirable method for the isolation of various brain products, and their ultimate organic analysis (2), to the investigations of Halliburton and Mott on the chemical pathology of hyperpyrexia (3), the rôle of cholin in epilepsy as shown by Donath (4), and the studies of Levene on the autodigestion of brain substance, (5). The underlying factors which cause the lecithin of the myelin sheath to split up into glycerophosphoric acid, stearic acid and cholin, the further analysis of these products, and finally the synthesis of lecithin from its decomposition products, afford a fruitful field for study. These latter problems are now the subject of investigation in this laboratory, but the results are not sufficiently ripe for publication.

2. Review of the Literature.

This will comprise only those communications which have appeared since the publication of my previous paper. One of the most important of recent contributions to the study of the cerebro-spinal fluid in all its aspects, has been the work of Sicard (6). The summary of its contents has already been touched

upon in my critical review (7). In the blood from the jugular veins of Cole's first case (8), one of acute alcoholic paralysis with a polyneuritic mental disorder and fatal after six weeks' duration, large quantities of cholin were found. Donath's communication I have been unable to consult, but in his paper on the treatment of general paralysis by means of artificial serum, he refers to the convulsive action of cholin when injected subdurally (9). The paper by Mott, Bury, Wilson, Sherrington, and Horsley is merely a summary of previous work on cholin, glycerophosphoric acid and the Marchi reaction (10).

Scott (11) has taken up the study of the micro-chemistry of nerve-cells. The author is led to conclude that there are at least three distinct nuclein compounds in nerve-cells, the Nissl granules, the basophilic substance covering the nucleolus and the oxyphile substance of the nucleus. Each of these bodies contains iron and phosphorus, the usual constituents of many nucleoproteids. These three compounds are genetically related, and a study of their developmental changes shows that they are derived from the chromatin of the nucleus of the germinating cells. This chromatin divides into two parts, each containing iron and phosphorus, but one is oxyphilic and remains in the nucleus, while the other is basophilic, diffuses into the cell-body and becomes a Nissl granule; but a portion of the nuclear chromatin remains unmodified about the nucleus.

Considerable work has been done by the French investigators on the action of lecithin and organic phosphorized compounds upon the animal organism and the development of the skeleton. The latest communication on this subject is by S. Hatai, on the effect of lecithin on the growth of the white rat (12). This observer, after a series of feeding and injection experiments with varying amounts of pure lecithin, arrives at the following conclusions: The rats which received the lecithin, either by injection or feeding, gained in body weight more rapidly than control rats which did not receive it, the gain being on the average sixty per cent. The relative weight of the central nervous system in the lecithin rats was normal and contained the same proportion of water and solids as the controls. Marinesco (13), on the lesions of the central nervous system following experimental hyperpyrexia and fever, deals exclusively with the anatomical side of the

question. Halliburton(14), on the contrary, working from a chemical standpoint, concludes that the physico-chemical cause of death from hyperpyrexia is due to the coagulation of cell-globulin (neuro-globulin), and when this constituent is thus coagulated, the protoplasm as such is destroyed. As the result of animal experiments and also of observations on human brain material taken as soon as possible after death, this coagulation temperature is shown to be 47° C., but temperatures as low as 42° C. will have the same effect if continued for a sufficient length of time. These changes are coincident with the chromatolytic phenomena in the nerve-cell, called by Marinesco coagulation necrosis.

Salkowski (15) has analyzed the cerebro-spinal fluid from a hydrocephalic child. The amount obtained was 1050 cc.; it was slightly cloudy, of alkaline reaction and having a specific gravity of 1006. A golden-yellow pigment was present, related to lutein. The proteid was in the form of serum albumin and serum globulin, and there was present a fermentative carbohydrate. In my paper on the cerebro-spinal fluid in hydrocephalus (16) particular attention was paid to the nature of the proteid and the reducing body, and also the physical chemistry of the fluid. I was fortunately able to secure an amount of fluid sufficient for a complete analysis (750 cc.). The molecular concentration, as established by the freezing-point method, was found to be greater that that of normal blood-serum; the pigment was a lipochrome analogous to the lutein of Kühne; the proteid was found to consist entirely of globulin, while the reducing body was definitely established to be dextrose, and not pyrocatechin, as claimed by Halliburton. This latter observation has also been corroborated by recent investigations of others.

Sollmann (17) has analyzed the cerebro-spinal fluid in five cases; in four he found it perfectly clear and colorless, in one there was a small sediment. The specific gravity ranged between 1.0137 and 1.0079. The reaction was slightly alkaline in all the cases and the freezing point ranged between —0.541° C. and —0.591° C. The total solids varied from 9.60 to 13.25, and the amount of ash from 6.00 to 7.23 per thousand parts. A reducing substance was present in two instances. Dircksen (18) claims that the cerebro-spinal fluid resembles the amniotic fluid, sweat

and lymph, by its large proportion of chlorides and the small amount of albumin and fibrin. In its normal state it contains neither agglutinins nor ferments. The chlorides are increased in those diseases where this material is retained in the organism; the reducing body is also increased in certain local meningitic processes and in diabetes. Concerning the latter condition, Schaefer's case is both instructive and valuable. There are large quantities of albumin in meningitis and uræmia. The normal freezing point is — 0.55° C.; this is increased in hydrocephalus, whether of local or general origin, and lowered in infectious diseases and uræmia. The normal fluid is not toxic, but becomes so in uræmia, tuberculous meningitis and grave forms of typhoid fever.

Thompson (19), in a monograph upon the spontaneous escape of cerebro-spinal fluid from the nose, gives the case of a woman, æt. 25, who suffered from almost continual dripping of the fluid from the nostril for two and one-half years, at times almost as much as 15 oz. in twenty-four hours. The patient in youth had suffered from headaches, and on various occasions, when the flow had ceased for a time, the headaches reappeared. There was no history of injury. The fluid was analyzed by Halliburton, and was found to be sterile, and containing pyrocatechin and a trace of globulin. The author gives the following differentiation between hydrorrhea and the escape of cerebro-spinal fluid from the nose:

Cerebro-spinal Fluid.	*Hydrorrhea.*
No mucin.	Pseudomucin.
Trace of globulin.	Proteid matter.
Pyrocatechin.	Sugar.

Sicard distinguishes between true and false nasal hydrorrhea; in one there is a hyper-secretion of the nasal mucous membrane, in the other there is an escape of cerebro-spinal fluid through the cribriform plate of the ethmoid.

Mott (20) in his recent paper, gives a slight modification of the previous method for the detection of cholin in the blood and cerebro-spinal fluid. It has the advantage of entirely eliminating platinum compounds of the potassium salts, but on the other hand it is less fitted for quantitative determinations than the previous methods. Furthermore, the possibility of contamination with

potassium salts is reduced to a minimum if the extraction with absolute alcohol is thorough and repeated several times. He finds that there is a proportional relationship between the presence of cholin in the blood and the amount of nervous tissue undergoing active degeneration. Cholin was found in the following fatal cases in which the blood was obtained shortly after death: three cases of beri-beri, two of tabo-paralysis ending in convulsive seizures, and one case each of acute alcoholic neuritis, combined sclerosis and Pott's disease. In three cases of disseminated sclerosis, two of amyotrophic lateral sclerosis, and one of focal myelitis, cholin was found in samples of the blood taken during life. The blood was also examined in fourteen cases of organic disease of the nervous system and in all of these cholin was found to be present in abnormal amounts and proportional to the extent and activity of the destruction of nervous tissue. Cholin and other bodies of the lecithin group were found in the blood of patients dying from beri-beri, and the blood taken during life from several cases of general paralysis, suffering with continuous epileptiform seizures, yielded considerable quantities of cholin. In other cases, where there were no fits, and where there was but little evidence of recent active degeneration, only comparatively small quantities were present. The author concludes that the test is of little value to decide whether a disease is organic or functional in nature, unless the organic disease be active at the time the blood is drawn. He also notes that other intermediate bodies between lecithin and cholin may exist in the blood and possess a toxic action.

3. Analyses of the Cerebro-Spinal Fluid in Various Mental Diseases.

The cases in this series number twenty-nine, and the abstracts of the clinical histories and autopsy findings are given rather fully. Detailed analyses of the cerebro-spinal fluid were made in the majority of the cases, and where the amounts secured permitted other data along special lines are given. The specific gravity was taken at 15° C. by means of an accurate urinometer. Cholin was tested for by the usual methods, and quantitated by filtering the last alcoholic extract into a weighed watch-glass. A few drops of 4% platinum chloride were added, evaporated first over

calcium chloride and then dried in an oven at 50° C. If the typical single and twin octahedral crystals were found, cholin was designated as present, the amount being the difference in weight between the original watch-glass and the mass of crystals therein. The platinum in this combination $(C_5 H_{14} NO Cl)_2$—$Pt Cl_4$ is 31.64%, from which the weight of the pure alkaloid is easily calculated. The freezing point was taken by Beckmann's apparatus. The proteid was estimated by precipitation with alcohol and filtering through a filter paper of known weight. The amount of reducing body was estimated by titration against standard Fehling's solution, and the results expressed in terms of dextrose. Lactic acid, urea, total solids and the amount and nature of the various salts were determined by the usual methods. All the quantities were expressed on the basis of 100 cc.

For complete analysis where only small amounts of cerebrospinal fluid are available, I would suggest the following procedure, which has been adopted in these cases. The reaction is first taken, and this followed by a determination of the freezing point. An aliquot portion of the fluid is then taken, the proteids are precipitated by alcohol and weighed, and the filtrate is used for cholin. Another smaller portion is used for the reducing body and lactic acid. The above data when made to include the amount, the time after death and the color, give a fairly complete analysis of the fluid, and are sufficient for all clinical investigation.

Case 1.—M. J. K., male, aged 67. Depressive hallucinatory delirium of three months' duration. The patient had been a steady drinker all his life, and two weeks before admission he had become confused and wandered away from home. During his stay in the hospital the mental picture was a varying one, from calm with a clear sensorium to episodes which showed marked confusion and exalted religious ideas. Physically the patient was rather weak and emaciated, the knee-jerks were absent and there was considerable arteriosclerosis. The urine showed a trace of albumin and a few casts. Once he went into a stuporous condition resembling uræmia. The emaciation gradually increased, slight fever supervened, he became delirious and disoriented, and, two days before death, there suddenly appeared continual twitchings of both arms, more marked on the right, and decided rigidity of the legs and neck muscles.

The autopsy showed both lungs to be riddled with tubercular nodules and cavities.

Brain weight, 1100 grms. Dura much thickened and strongly adherent; pia œdematous and hazy; right olfactory bulb partially atrophied;

fourth ventricle free from granulations. The paraffin-Nissl sections of the frontals and paracentrals showed the axonal reaction of the large pyramidal cells, with pigmentation.

Analysis of the Cerebro-spinal Fluid.

Amount	90 cc.
Time after death	1 hour.
Color	clear
Proteid	large amount.
Cholin	present.

CASE 2.—W. T. C., male, aged 44. General paralysis, tabetic form, with many absurd somatic ideas, of three years' duration. Death from bronchopneumonia and exhaustion.

Brain weight, 1325 grms. Vessels thickened and injected. Convolutions flattened, frontal lobes firmly adherent. Softening of the frontal poles. Pia hazy and very œdematous; distinct granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount	150 cc.
Time after death	1 hour.
Color	blood-tinged (contamination).
Cholin	present.

Cholin was also found in 10 grms. of brain tissue taken from the first left frontal convolution and in 5 grms. of the cord taken from the region of the seventh cervical segment.

CASE 3.—M. J. D., male, aged 72. Senile paranoiac condition, with marked depressive traits, of two years' duration. Onset with a peculiar feeling of weakness in the abdomen, later an indefinite suspicion that his wife's relatives were trying to acquire his property; he became seclusive, planned to kill himself, this leading to his commitment. Here at the hospital, he was restless, indifferent, frequently talking of his "nonsense ideas" at home, yet without any true insight. Bradycardia, bigeminal pulse, hypertrophy of left ventricle, mitral systolic murmur, transmitted to the axilla. On November 14, 1902, gangrene of the right foot set in and there was considerable fever. The condition improved but little, however, even under surgical treatment. The urine contained a large trace of albumin, increased indican and waxy casts. The patient gradually became stuporous, and there was ptosis of the right eyelid. At first there were a few twitchings of the fingers and forearm muscles, with rigidity of the neck and elbows. The twitchings gradually increased in intensity and became especially marked in the left arm. Death from lobar pneumonia.

Autopsy.—Brain weight, 1280 grams. Dura thickened and strongly adherent. Pia moderately injected. Convolutions flattened and atrophic over parietal and frontal regions. Basal vessels much thickened. No granu-

lations in the fourth ventricle. The pulmonary artery was guarded by a valve containing four cusps. The axonal reaction was present in a few of the large pyramidal cells of the frontals and paracentrals.

Analysis of the Cerebro-spinal Fluid.

Amount .....50 cc.
Time after death.....19 hours.
Color .....blood-tinged (contamination).
Cholin .....absent.

Cholin was also absent in 10 grms. of the brain substance taken from the first left frontal convolution and from the cord segments from the first to the fourth cervical.

CASE 4.—F. W. P., male, aged 75. Senile organic dementia of six months' duration. Sudden onset of paralysis of the right side of the face with right brachial monoplegia. Probable right hemianopsia (difficult to determine on account of the demented condition). Continual tremor of the left arm. Double Babinski reflex. Death from hypostatic pneumonia, ten days after the onset of the above symptoms.

Brain weight, 1315 grms. Dura much thickened and very adherent. Basal vessels thickened. No granulations of the fourth ventricle. Convolutions flat and broad. Tumor the size of a hazel-nut in the splenium of the corpus callosum.

Analysis of the Cerebro-spinal Fluid.

Time after death.....2 hours.
Amount .....45 cc.
Color .....blood-tinged (contamination).
Cholin.....present in large amount.

Many large crystals of the double platinum salt of cholin were also found in 10 grms. of tissue taken from the first left frontal convolution and from a cord segment comprising the eighth and ninth thoracic.

CASE 5.—G. H., male aged 72. Senile dementia of four years' duration. Marked confusion and delirium. Death from hypostatic pneumonia and pulmonary gangrene.

Brain weight, 1200 grms. Pia hazy, dura much thickened. Marked atrophy of the second left temporal convolution. Basal vessels much thickened. No granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount .....60 cc.
Time after death.....1 hour.
Color .....clear.
Cholin.....a few crystals present.

CASE 6.—P. K., male, aged 45. General paralysis (tabetic form) of two years' duration. Death from lobar pneumonia.

Brain weight, 1490 grms. Moderate œdema, substance fairly firm, a few fine granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount .....75 cc.
Time after death.....9 hours.
Color .....blood-tinged (contamination).
Cholin .....present.
Cholin (as platinum salt per 100 cc.).....0.480 grm.

Cholin was also present in 10 grms. of the first left frontal convolution, in 5 grms. of C_2 to C_3 and in two spinal ganglia, L L 3 and R L 3.

CASE 7.—M. McD., male, aged 33. Alcoholic depressive hallucinosis of ten months' duration. Pulmonary tuberculosis with sudden death. No autopsy.

Analysis of the Cerebro-spinal Fluid.

Amount .....70 cc.
Time after death.....2 hours.
Color .....clear.
Reaction .....slightly acid.
Specific gravity .....1006.
Lactic acid.....present (trace).
Reducing body...present. (Reduction of Fehling's solution. The osazon crystallized in fine, radiating, yellow needles.)
Cholin .....absent.
Freezing point .....($\triangle$) = —0.5° C.
Total proteid.....= 0.124 grm. in 100 cc. (0.124%).

CASE 8.—L. M., female, aged 62. Melancholia of four years' duration. Death from carcinoma of the uterus.

Brain weight, 1250 grms., soft over the post-central regions; atrophic area in the occipital lobe.

Analysis of the Cerebro-spinal Fluid.

Amount .....37 cc.
Color .....clear.
Time after death .....9 hours.
Reaction .....acid.
Lactic acid.....strong reaction.
Reducing body .....absent.
Freezing point.....($\triangle$) = —0.6° C.
Total proteid.....= 0.180 grm. in 100 cc. (0.18%).
Cholin .....present.
Cholin (as platinum salt).....= 0.368 grm. per 100 cc.

CASE 9.—J. McA., male, aged 40. General paralysis of five years' duration. Death from pulmonary tuberculosis.

Brain weight, 1029 grms. Pia thickened and very œdematous; convolutions atrophic in the frontal and central regions; numerous granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Time after death	7 hours.
Amount	25 cc.
Color	clear.
Reaction	acid.
Lactic acid	present.
Reducing body	absent.
Freezing point	(Δ) = —0.6° C.
Total proteid	= 0.130 grm. in 100 cc. (.13%).
Cholin	present.
Cholin (as platinum salt)	= 0.315 grm. in 100 cc.

Case 10.—F. de P., male, aged 25. Alcoholic delirium with polyneuritis. Duration one month. For two years the patient had used alcoholic liquors to excess. About a week before admission to the hospital he began to show failing memory, became pallid and weak in appearance, was restless and had auditory and visual hallucinations. On entrance to the hospital, there was some fever and he was quite weak. Knee-jerks and kneecap reflexes absent; tremor of hands and tongue; pupils equal and react promptly; calves extremely tender to pressure, and the calf muscles are very flabby. Unable to distinguish between heat and cold on any part of the legs. "Drop" of left foot. Tenderness over intercostal, anterior crural, tibial and popliteal nerves on both sides. The patient gradually grew weaker, the fever continued, the sciatics, the peroneals and the median nerves became involved, Cheyne-Stokes respiration set in, there was a sudaminous eruption over the upper part of the body, and some rigidity of the arms. He became totally unconscious and died, three weeks after admission, of multiple neuritis. The autopsy was performed twenty-five minutes after death.

Brain weight, 1380 grms. No œdema. Slight haziness along the lines of the vessels. The fourth ventricle was free from granulations. The Nissl specimens of the frontals and paracentrals showed some acute alteration of the large pyramids, but no axonal reaction.

Analysis of the Cerebro-spinal Fluid.

Amount	30 cc.
Time after death	10 minutes.
Color	cloudy yellow.
Reaction	slightly acid.
Lactic acid	trace.
Reducing body	marked reaction.
Freezing point	(Δ) = —0.7° C.
Total proteid	0.364 grm. in 100 cc. (0.364%).
Cholin	present.
Cholin (as platinum salt)	0.588 grm. per 100 cc.

There was no cholin in the blood. Ten grms. of brain tissue, taken from the first left frontal convolution, yielded many large and fine typical

crystals. Cholin was also obtained from the following peripheral nerves, right and left sciatic, median and anterior crural. The following shows the relation between the presence of cholin and the extent of the Marchi reaction, in the brain, cord and peripheral nerves:

Tissue.	Cholin.	Marchi Reaction.
L F 1	Many large and fine crystals.	A few fibers, fairly well marked.
L P C L	" " "	" " " " "
R P C L	" " "	" " " " "
R F 1	" " "	" " " " "
Medulla	" " "	" " " " "
C-1		Many fibers, well marked.
Th-11		" " " "
L-2		Numerous fibers, well marked.
L. Ant. crural nerve	A few typical crystals.	A few fibers.
R. Ant. crural nerve	" " "	(Nerve not taken for Marchi.)
Right median nerve	" " "	A few fibers.
Left median nerve	" " "	" "
Right sciatic nerve	" " "	" "
Left sciatic nerve	" " "	" "
Left int. popliteal nerve		Many degenerated fibers.
Right int. popliteal nerve		" " "

CASE 11.—J. M., male, aged 65. General paralysis with double optic atrophy of two years' duration. Death from hypostatic pneumonia.

Brain weight, 1280 grms. Thickening of the dura; pia hazy and œdematous; atrophic areas in the left frontal region; olfactory bulbs tightly bound down. No granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount	70 cc.
Time after death	9 hours.
Color	blood-tinged (contamination).
Cholin	present.
Cholin (as platinum salt)	0.158 grm. in 100 cc.

CASE. 12.—H. M., male, aged 41. General paralysis. Ataxic demented form of five years' duration. Death from hypostatic pneumonia. No autopsy.

Analysis of the Cerebro-spinal Fluid.

Amount	33 cc.
Time after death	4 hours.
Color	clear, lemon-yellow.
Reaction	slightly acid.
Lactic acid	present.
Reducing body	absent.
Freezing point	$(\triangle) = -0.65°$ C.
Total proteid	= 0.333 grm. per 100 cc. (0.333%).
Cholin	present.
Cholin (as platinum salt)	0.489 grm. in 100 cc.

CASE 13.—H. D., female, aged 77. Senile dementia of five years' duration. Aortic stenosis. For eleven days before death there were noted progressive weakness and emaciation, coarse spasmodic twitching of the face and extremities, rigidity of arms and elevation of temperature. Death was due to broncho-pneumonia. There was no autopsy.

Analysis of the Cerebro-spinal Fluid.

Amount .....25 cc.
Time after death.....45 minutes.
Color .....clear.
Reaction .....slightly acid.
Lactic acid .....trace.
Reducing body.....present (marked).
Freezing point.....(Δ) = —0.75° C.
Total proteid.....0.185 grm. in 100 cc.
Cholin .....present
Cholin (as platinum salt).....0.480 grm. in 100 cc.

CASE 14.—J. K., male, aged 34. General paralysis of seven years' duration. Death from cerebral hemorrhage and lobar pneumonia.

Brain weight, 1320 grms. Subdural hemorrhagic membrane over the posterior part of the right hemisphere. Frontal convolutions, paracentrals and cunei flattened and atrophic. Granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount .....80 cc.
Time after death.....9 hours.
Color .....blood-tinged.
Cholin .....present.
Cholin (as platinum salt) .....0.393 grm. in 100 cc.

CASE 15.—J. M., male, aged 51. General paralysis, demented ataxic form, of one year's duration. Beginning double optic atrophy. Death from heart failure.

Brain weight, 1440 grms. Considerable pial œdema; frontal lobes adherent; cisterna thickened and hazy; granulations in the fourth ventricle. There was grayness of the posterior columns of the cord, most marked in the lumbar region.

Analysis of the Cerebro-spinal Fluid.

The fluid was obtained by lumbar puncture ten minutes after death, and was still warm. The pressure was considerable, as it flowed very rapidly, at times in almost a steady stream. After standing for about fifteen minutes, a few fine threads formed in the liquid (fibrin?).

Amount .....140 cc.
Color.....clear, with a very slight yellow tinge.
Reaction.....very slightly acid.

Lactic acid .....trace.
Specific gravity .....1009.
Freezing point.....(∇) = —0.65° C.
Cholin .....present.
Cholin (as platinum salt).....0.374 grm. in 100 cc.
Total proteid.....0.132 grm. in 100 cc. (0.132%).
Nucleo-proteid .....present.
Serum albumin .....trace.
Serum globulin.....large amount.
Albumose .....absent.
Cholin (as alkaloid).....0.1183 grm. in 100 cc. (.118%).
Peptone .....absent.
Sodium chloride .....present.
Total NaCl.....0.8 grm. in 100 cc. (.8%).
$\frac{\Delta}{\text{NaCl\%}}$ .....0.819.
Cholesterin .....absent.
Urea .....present.
Total urea.....0.150 grm. in 100 cc. (0.15%).
Total solids.....3.420 grms. in 100 cc.
Reducing body...well-marked reaction with Fehling's solution.

After removal of the proteid by heat and acetic acid, the proteid free solution responded to the following reactions:

Fehling's solution.....marked reduction.
Nylander's solution .....negative.
Molisch's reaction .....negative.
Fermentation test .....negative.
Ferric chloride.....slight green tinge.
Sodium hydrate (with heating).....slight brown color.
Phenylhydrazin test—a small number of fine, long, yellow, needle-like crystals arranged in sheaves, but too few in amount to determine the melting point.

The amount of this reducing body, in terms of dextrose, was 0.0555 grm. in 100 cc.

TABLE OF SUBSTANCES (ON BASIS OF 100 CC.).

Total Solids.	*Organic Substances.*		*Inorganic Salts.*
3.420 grms.	Reducing body....	0.0555 grm.	2.9642 grms.
	Cholin	0.1183 grm.	(of which .8 grm.
	Proteid	0.132 grm.	is NaCl).
	Urea	0.150 grm.	
		0.4558 grm.	

CASE 16.—C. McL., male, aged 67. General paralysis of two years' duration. Death from hypostatic pneumonia.

Brain weight, 1290 grms. Frontal poles very soft; atrophy of frontal and precentral convolutions. Granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount	35 cc.
Color	blood-tinged (contamination).
Time after death	10 hours.
Cholin	present.
Cholin (as platinum salt)	0.450 grm. in 100 cc.

CASE 17.—E. D., female, aged 63. Traumatic organic dementia of one month's duration. Three weeks before admission to the hospital, the patient is said to have sustained a fall on the ice. She was unconscious, was found stupid, and paralyzed on both sides and rapidly became confused and delirious. In the hospital she was stuporous, did not speak; there was Cheyne-Stokes respiration, no motor paralysis of the limbs, but a slight drooping of the left corner of the mouth. At first there was no disturbance of vision, but later right hemianopsia was made out. She developed left parotitis, rapidly failed and died of lobar pneumonia.

Brain weight, 1420 grms. Dura much adherent, especially in the occipital region. The left hemisphere appears fuller than the right. No granulations in the fourth ventricle. A longitudinal section of the brain shows a tumor mass, irregular in outline, through the center of the corpus callosum, and extending upwards into the cortex for about 1.5 cm.

Analysis of the Cerebro-spinal Fluid.

Amount	25 cc.
Color	deep, cloudy yellow.
Time after death	9 hours.
Reaction	slightly acid.
Lactic acid	present.
Freezing point	$(\Delta) = -0.8°$ C.
Reducing body	absent.
Total proteid	0.270 grm. in 100 cc. (.27%).
Cholin	present.
Cholin (as platinum salt)	0.580 grm. in 100 cc.

CASE 18.—M. R., female, aged 69. Alcoholic senile deterioration of long duration. Nine days before death, there was an apoplectic seizure with almost complete motor paralysis of the left side.

Brain weight, 1130 grms. Softer than normal; several atrophic areas over the vertex; no granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount	20 cc.
Time after death	8 hours.
Color	clear and slightly yellow.
Reaction	acid.

Lactic acid	strong reaction.
Urea	present.
Total urea	0.3 grms. per 100 cc. (0.3%).
Freezing point	($\triangle$) = —0.72° C.
Reducing body	marked reaction.
Total proteid	0.390 grm. in 100 cc. (0.39%).
Cholin	present.
Cholin (as platinum salt)	0.780 grm. in 100 cc.

CASE 19.—U. H., male, aged 64. General paralysis of two years' duration. Death from lobar pneumonia.

Brain weight, 930 grms. Very œdematous; pia thickened. The convolutions are much atrophied, especially in the frontal lobes, but becoming less marked as the occipital region is approached. The post-central convolution on the right is almost entirely shrunk away. No granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

The fluid was obtained by lumbar puncture, seven hours after death. It flowed very rapidly and with considerable pressure. It contained a few fine threads, probably fibrin.

Amount	125 cc.
Color	clear, yellow.
Reaction	acid.
Lactic acid	present.
Specific gravity	1012.
Freezing point	($\triangle$) = —0.9° C.
Nucleo-proteid	present.
Serum albumin	present.
Serum globulin	present.
Albumose	absent.
Peptone	absent.
Chlorides	present.
Phosphates	absent.
Sulphates	trace.
Cholesterin	absent.
Urea	present.
Total urea	0.5 grm. in 100 cc. (0.5%).
Total solids	2.160 grms. in 100 cc.
Total NaCl	1.240 grms. in 100 cc.
$\frac{\triangle}{\text{NaCl\%}}$	0.726.
Cholin	present.
Cholin (as alkaloid)	0.1771 grm. in 100 cc. (0.177%).
Cholin (as platinum salt	0.560 grm. in 100 cc.
Total proteid	0.142 grm. in 100 cc. (0.142%).
Reducing body	present. (Reaction well marked.)

After removal of the proteid by heat and acetic acid, the fluid responded to the following tests:

Fehling's solution.....marked reduction.
Nylander's solution .....negative.
Fermentation test .....positive.
Molisch's test .....negative.
Ferric chloride .....negative.
Sodium hydrate (with warming).....marked brownish color.
Phenylhydrazin test—a few yellow needles resembling phenyl-glucosozan. The amount of this reducing body (in terms of dextrose) was .0476 grm. per 100 cc.

Cholin was also present from an atrophic area taken from the first left frontal convolution.

TABLE OF SUBSTANCES (ON BASIS OF 100 CC.).

Total Solids.	*Organic Substances.*	*Inorganic Salts.*
2.160 grms.	Reducing body.....0.476 grm.	1.2933 grms.
	Cholin .....0.1771 grm.	(of which 1.24 grms.
	Proteid .....0.142 grm.	is NaCl).
	Urea .....0.500 grm.	
	0.8667 grm.	

CASE 20.—J. T., male, aged 51. Melancholia of two years' duration. Death from exhaustion and hypostatic pneumonia. Extreme emaciation.

Brain weight, 1460 grms. Substance flabby; no granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount .....30 cc.
Time after death.....9 hours.
Color .....blood-tinged (contamination).
Cholin .....absent.

CASE 21.—G. McW., male, aged 40. General paralysis of four years' duration. Death from pulmonary tuberculosis.

Brain weight, 1290 grms. Pia hazy, substance flabby; frontal lobes tightly adherent. No granulations in the fourth ventricle. There is a 2×1.5 cm. cyst on the third left temporal convolution.

Analysis of the Cerebro-spinal Fluid.

Amount .....15 cc.
Time after death.....12 hours.
Color .....clear, yellow.
Reaction .....slightly acid.
Lactic acid .....trace.
Reducing body .....absent.
Total proteid .....0.180 grm. in 160 cc. (.18%).
Cholin .....present.
Cholin (as platinum salt).....0.430 grm. in 100 cc.

CASE 22.—G. Le M., male, aged 79. Senile dementia of three years' duration. Death from hypostatic pneumonia.

Brain weight, 1275 grms. Pia œdematous; substance flabby over frontal poles; basal vessels much thickened.

Analysis of the Cerebro-spinal Fluid.

Amount	18 cc.
Time after death	6 hours.
Color	clear, yellow.
Reaction	slightly acid.
Lactic acid	trace.
Freezing point	$(\triangle) = -0.75°$ C.
Reducing body	slight reaction.
Total proteid	0.1188 grm. in 100 cc. (11%).
Cholin	absent.

CASE 23.—J. B., male, aged 53. General paralysis of three years' duration. Death from hypostatic pneumonia. No autopsy.

Analysis of the Cerebro-spinal Fluid.

Amount	25 cc.
Color	clear, yellow.
Time after death	3 hours.
Reaction	slightly alkaline.
Lactic acid	absent.
Reducing body	present and well marked.
Freezing point	$(\triangle) = -0.7°$ C.
Urea	present.
Total urea	0.2 grm. per 100 cc. (.2%).
Total proteid	0.405 grm. per 100 cc. (.4%).
Cholin	present.
Cholin (as platinum salt)	0.520 grm. in 100 cc.

CASE 24.—W. K., male, aged 42. General paralysis of one year's duration. Death from broncho-pneumonia.

Brain weight, 1430 grms. Pia hazy; marked œdema; convolutions of frontal lobes atrophic; granulations in the fourth ventricle. The stomach contained about 500 cc. of a dark, reddish-brown fluid, which gave all the tests for bile. The gall-bladder was distended with the same fluid.

Analysis of the Cerebro-spinal Fluid.

Amount	40 cc.
Time after death	10 minutes.
Color	cloudy, yellow.
Reaction	slightly acid.
Lactic acid	trace.
Reducing body	marked reaction.
Specific gravity	1012.

Bile pigment	present.
Urea	present.
Total urea	0.1 grm. in 100 cc. (0.1%).
Sodium chloride	.9 grm. in 100 cc.
Freezing point	$(\triangle) = -0.6°$ C.
$\frac{\triangle}{NaCl\%}$	0.666.
Total proteid	0.252 grm. in 100 cc. (.25%).
Cholin	present.
Cholin (as platinum salt)	0.664 grm. in 100 cc.

CASE 25.—K. B., male, aged 36. General paralysis of two years' duration. Headache, tremor and weakness of the right arm, epileptiform convulsions confined mostly to the left side. Death from exhaustion and lobar pneumonia.

Brain weight, 1325 grms. Considerably injected, frontals firmly adherent; right hemisphere smaller in every way than the left (hemi-atrophy); temporals firmly adherent; granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount	30 cc.
Time after death	8 hours.
Color	clear.
Reaction	strongly acid.
Lactic acid	marked reaction.
Reducing body	absent.
Total proteid	0.308 grm. in 100 cc. (0.3%).
Cholin	present.
Cholin (as platinum salt)	0.684 grm. in 100 cc.

CASE 26.—G. R., male, aged 62. General paralysis, tabetic form. Death from lobar pneumonia.

Brain weight, 1380 grms. Dura thickened, marked pial œdema; the frontal convolutions have pitted, atrophic areas. Basal vessels thickened. Granulations in fourth ventricle. There is a ½-cm. organized clot at the junction of the basilar with the vertebrals.

Analysis of the Cerebro-spinal Fluid.

Amount	60 cc.
Time after death	45 minutes.
Color	cloudy, yellow.
Specific gravity	1009.
Reaction	alkaline.
Lactic acid	absent.
Reducing body	absent.
Urea	0.1 grm. in 100 cc. (0.1%).
Freezing point	$(\triangle) = -0.55°$ C.
Sodium chloride	0.8 grm. in 100 cc. (0.8%).

$\frac{\Delta}{NaCl\%}$ .....0.689.
Total proteid.....0.212 grm. in 100 cc. (0.2%).
Cholin .....present.
Cholin (as platinum salt).....0.360 grm. in 100 cc.

CASE 27.—H. L. M., female, aged 30. Polyneuritic psychosis with multiple neuritis of four months' duration. No autopsy.

Analysis of the Cerebro-spinal Fluid.

Amount .....10 cc.
Time after death.....8 hours.
Color .....blood-tinged (contamination).
Cholin .....present.
Cholin (as platinum salt).....0.520 grm. per 100 cc.

CASE 28.—W. W. P., male, aged 65. Manic-depressive insanity, circular form, third attack of three years' duration. Transitory diplopia one year before death, followed by complete left hemiplegia a couple of months later. Death from tubercular pleurisy.

Brain weight, 1360 grms. Dura very adherent; right pre-central convolution a little depressed; basal vessels much thickened; no granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount .....10 cc.
Time after death.....14 hours.
Color .....cloudy, yellow.
Cholin .....present.
Cholin (as platinum salt).....0.630 grm. in 100 cc.

CASE 29.—A. V. B., male, aged 35. General paralysis, tabetic form, of nine years' duration. Sudden death from heart failure.

Brain weight, 1260 grms. Substance extremely soft; convolutions flattened and sulci shallow; granulations in the fourth ventricle.

Analysis of the Cerebro-spinal Fluid.

Amount .....85 cc.
Time after death.....4 hours.
Color .....clear.
Reaction .....acid.
Lactic acid .....present.
Specific gravity .....1009.
Reducing body .....absent.
Freezing point.....(Δ) = −0.45° C.
Urea .....present.
Total urea.....0.1 grm. in 100 cc. (0.1%).
Total proteid.....0.156 grm. in 100 cc. (0.15%).
Cholin .....present.
Cholin (as platinum salt).....0.396 grm. in 100 cc.

4. Summary of Results.

The amounts of fluid varied from 10 cc. to 15 cc., the highest figures being found in general paralysis. In this disease, out of fifteen cases, it was found to exceed 50 cc. in eight, and in only one was it below 25 cc. This of course finds its explanation in the excessive pial œdema. The cases in which only small amounts were obtained can be explained on the possibility that perhaps by the method of lumbar puncture only a portion of a fluid escaped. In two cases of alcoholic neuritis and one of chronic alcoholic deterioration, the amount was small; in two others of the depressive hallucinatory type, 70 cc. and 90 cc. were respectively obtained. In five senile cases, including the organic, demented, and paranoiac forms, the amounts varied from 18 cc. to 60 cc. In two of melancholia it was 33 cc. and 37 cc.; in a traumatic organic case, 25 cc., but in a case of manic-depressive insanity with a terminal hemiplegia, only 10 cc. were obtained.

The fluid was secured at times varying from 10 minutes to 19 hours after death. In all, it was clear or of a slightly yellow tinge (except for an occasional accidental contamination with blood). In one case, however, (24) bile pigment was present, being due to absorption from the intestinal canal. Only twice was the reaction alkaline and lactic acid absent; in all the others, the reaction varied from slightly to strongly acid, this being due in every instance to lactic acid in varying amounts. The total acidity, by titration with $\frac{N}{10}$ NaOH was not determined in any of the tests. In only six cases was there obtained sufficient fluid for the specific gravity. In one of these it was low (1006), in the other five, cases of general paralysis, it was 1009 in three and 1012 in two.

A reducing substance was present in nine instances. In all excepting three, Fehling's solution was the only test used, after removal of the proteid by acetic acid and heat. In case 7, the osazon crystallized in fine radiating needles; in cases 15 and 19, the body was subjected to a more elaborate study. Nylander's, Molisch's and the ferric chloride reaction were negative; a brown color was obtained by warming with strong sodium hydrate, and on fermentation with yeast in one case only was there a production of carbon dioxide. The osazon exactly resembled phenylglucosazon. The amount of this reducing substance, ex-

TABLE OF RESUL

No. of Case.	Diagnosis.	Amt.	Color.	Time after Death.	Spec. Grav.	Reaction.	Lactic Acid.	Reducing Body.	Freezing Point
I.	Depressive Hallucinatory Delirium.	90 cc.	Clear.	1 hour.	....	.....	.....	.....	....
II.	General Paralysis (tabetic form).	150 cc.	Blood-tinged	1 hour.	...	.....	... ..	.....	....
III.	Senile paranoic condition.	50 cc.	Blood-tinged.	19 hours.	....	.....	.....	.....	....
IV.	Senile Organic Dementia.	45 cc.	Blood-tinged	2 hours.	....	.....	.....	.....	....
V.	Senile Dementia.	60 cc.	Clear.	1 hour.	....	.....	.....	.....	....
VI.	General Paralysis (tabetic form).	75 cc.	Blood-tinged.	9 hours.	....	.....	.....	.....	.. ..
VII.	Alcoholic Depressive Hallucinosis.	70 cc.	Clear.	2 hours.	1006	Sl. acid.	Trace.	Present.	−0.5°
VIII.	Melancholia.	37 cc.	Clear.	9 hours.	....	Acid.	Strong reaction.	Absent.	−0.6°
IX.	General Paralysis.	25 cc.	Clear.	7 hours.	....	Acid.	Present.	Absent.	−0.6°
X.	Alcoholic Delirium (polyneuritis).	30 cc.	Cloudy, yellow.	10 mins.	....	Sl. acid.	Trace.	Marked reaction.	−0.7°
XI.	General Paralysis.	70 cc.	Blood-tinged.	9 hours.	....	.....	.....	.....	....
XII.	General Paralysis (tabetic form).	33 cc.	Clear, yellow.	4 hours.	....	Sl. acid.	Present.	Absent.	−0.65
XIII.	Senile Dementia.	25 cc.	Clear.	45 mins.	....	Sl. acid.	Trace.	Present.	−0.75
XIV.	General Paralysis.	80 cc.	Blood-tinged.	9 hours.	....	.....	.....	.....	....
XV.	General Paralysis (tabetic form).	140 cc.	Clear.	10 mins.	1009	Sl. acid.	Trace.	Present.	−0.65
XVI.	General Paralysis.	35 cc.	Blood-tinged.	10 hours.	....	.....	.....	.....	....
XVII.	Organic Dementia (traumatic).	25 cc.	Cloudy, yellow.	9 hours.	....	Sl. acid.	Present.	Absent.	−0.8°
XVIII.	Alcoholic Senile Deterioration.	20 cc.	Clear.	8 hours.	....	Acid.	Strong reaction.	Marked.	−0.72
XIX.	General Paralysis.	125 cc.	Clear, yellow.	7 hours.	1012	Acid.	Present.	Present.	−0.9°
XX.	Melancholia.	30 cc.	Blood-tinged.	9 hours.	....	.....	.....	.....	....
XXI.	General Paralysis.	15 cc.	Clear, yellow.	12 hours.	....	Sl. acid.	Trace.	Absent.	....
XXII.	Senile Dementia.	18 cc.	Clear, yellow.	6 hours.	....	Sl. acid.	Trace.	Slight.	−0.75
XXIII.	General Paralysis.	25 cc.	Clear, yellow.	3 hours.	....	Sl. alk.	Absent.	Present.	−0.7°
XXIV.	General Paralysis.	40 cc.	Cloudy, yellow.	10 mins.	1012	Sl. acid.	Trace.	Marked.	−0.6°
XXV.	General Paralysis.	30 cc.	Clear.	8 hours.	....	Strongly acid.	Marked.	Absent.	....
XXVI.	General Paralysis (tabetic form).	60 cc.	Cloudy, yellow.	45 mins.	1009	Alkaline.	Absent.	Absent.	−0.55
XXVII.	Polyneuritic Psychosis.	10 cc.	Blood-tinged.	8 hours.	....	.....	.....	.....	....
XXVIII.	Manic-Depressive Insanity.	10 cc.	Blood-tinged.	14 hours.	....	.....	.....	.....	....
XXIX.	General Paralysis (tabetic form).	85 cc.	Clear.	4 hours.	1009	Acid.	Present.	Absent.	−0.45

BLE OF RESULTS.

educing Body.	Freezing Point.	Proteid.	Cholin.	Na Cl.	$\frac{\Delta}{\text{Na Cl's.}}$	Urea.	Remarks.
.....	.....	Large amount	Present.	.....	.....	.....	Central neuritis.
.....	.....	.....	Present.	.....	.....	.....	Cholin also found in brain and cord.
.....	.....	.....	Absent.	.....	.....	.....	Central neuritis.
.....	.....	.....	Present.	.....	.....	.....	Cholin also found in brain and cord.
.....	.....	.....	Small amount.	.....	.....	.....	
.....	.....	.....	0.480 g.	.....	.....	.....	Cholin also found in brain, cord and ganglia.
resent.	−0.5° C.	0.124 g.	Absent.	.....	.....	.....	
bsent.	−0.6° C.	0.180 g.	0.368 g.	.....	.....	.....	
bsent.	−0.6° C.	0.130 g.	0.315 g.	.....	.....	.....	
Iarked eaction.	−0.7° C.	0.364 g.	0.588 g.	.....	.....	.....	Cholin also found in brain and peripheral nerves, none in blood.
.....	.....	.....	0.158 g.	.....	.....	.....	Double optic atrophy.
bsent.	−0.65° C.	0.333 g.	0.489 g.	.....	.....	.....	
resent.	−0.75° C.	0.185 g.	0.480 g.	.....	.....	.....	Central neuritis.
.....	.....	.....	0.393 g.	.....	.....	.....	
resent.	−0.65° C.	0.132 g.	0.374 g.	0.8 g.	0.819	0.150 g.	Complete analysis.
.....	.....	.....	0.450 g.	.....	.....	.....	
bsent.	−0.8° C.	0.270 g.	0.580 g.	.....	.....	.....	
Iarked.	−0.72° C.	0.390 g.	0.780 g.	.....	.....	0.3 g.	
resent.	−0.9° C.	0.142 g.	0.560 g.	1.240 g.	0.726	0.5 g.	Complete analysis.
.....	.....	.....	Absent.	.....	.....	.....	
bsent.	.....	0.180 g.	0.430 g.	.....	.....	.....	
light.	−0.75° C.	0.1188 g.	Absent.	.....	.....	.....	
resent.	−0.7° C.	0.405 g.	0.520 g.	.....	.....	0.2 g.	
Iarked.	−0.6° C.	0.252 g.	0.664 g.	0.9 g.	0.686 g.	0.1 g.	Bile pigment present.
bsent.	.....	0.308 g.	0.684 g.	.....	.....	.....	
bsent.	−0.55° C.	0.212 g.	0.360 g.	0.8 g.	0.689 g.	0.1 g.	
.....	.....	.....	0.520 gr.	.....	.....	.....	
.....	.....	.....	0.630 g.	.....	.....	.....	Left hemiplegia.
bsent.	−0.45° C.	0.156 g.	0.396 g.	.....	.....	0.1 g.	

pressed in terms of dextrose, was 0.0555 grm. and 0.0476 grm. per 100 cc. respectively. The above reactions, in conjunction with a further study of this body in my case of hydrocephalus, in which the melting point of the osazon was determined, and care taken to exclude the presence of pentoses, have led me to conclude that the substance is dextrose, and the reaction is not due to pyrocatechin or to any derivatives of the cerebrins. The alleged disappearance of the reducing power of the cerebro-spinal fluid, when some time elapses between the time of death and that of testing, was not observed in this series, both positive and negative results being obtained with various fluids, whose time of removal almost exactly coincided. This conclusion as to the nature of the reducing body has been verified by other investigators.

Urea was estimated in seven cases, and varied from 0.1 grm. to 0.5 grm. per 100 cc. of fluid. It is of no pathological significance, and although the French authors point out its almost exclusive appearance in uremic conditions, yet a large amount of the substance was found in a case of hydrocephalus, where the character of the fluid approaches nearest to the type of a normal secretion.

The freezing point was determined in fifteen cases, and in only three of these was it less that in normal blood serum. The results of this method, as a measure of the osmotic pressure and molecular concentration, places the cerebro-spinal fluid in the class of a true secretion, and not merely a blood transudate or filtration. The depression of the freezing point bears no relation to the amount of proteid present, for cases are recorded above in which the freezing point was lower and the amount of proteid material smaller, than in those fluids where the freezing point approached nearest to that of normal blood serum. On the other hand there is some parallel with the specific gravity, for those cases with the highest specific gravity have the lowest freezing point, although both are independent of the amount of proteid present. For example, in case 19, the freezing point was —0.9 C., the specific gravity 1012 and the proteid content 0.142 grm. per 100 cc.; whereas, in case 29, the freezing point was —0.45° C., the specific gravity was 1009, and the total proteid 0.156 grm. per 100 cc. This leads us to other conclusions and

the apparent discrepancy becomes intelligible if we remember that the osmotic pressure of a solution as determined by the freezing point method, is dependent upon the number of dissociated ions which the solution contains. The proteid molecule, being so large and complex, fails of dissociation, and therefore is ineffective in increasing the osmotic pressure. A ready explanation is found if we glance at those cases in which the total inorganic salts were determined, with special relation to the chlorides, which, although estimated as sodium chloride, is also in combination with the other univalent element, potassium, in the relation, Na : K :: 8 : 1 or 10 : 1. This is best illustrated by the following short table:

Case.	NaCl.	Freezing Point (Δ).	$\frac{\Delta}{NaCl\%}$
15	0.8 grm.	—0.65° C.	0.819
19	1.240 grms.	—0.9° C.	0.726
24	0.9 grm.	—0.6° C.	0.666
26	0.8 grm.	—0.55° C.	0.689

The above table readily shows the relation of the freezing point depression to the amount of sodium chloride, and is ample proof of its dependence upon the amount of this salt present, which as I have shown, comprises the greatest amount of the salt of the total solids, and is, therefore, most strongly dissociated. All this of course would be changed if the organic substances present were incinerated, because substances would be thereby formed by the process of heating, which, if dissolved in water in amount equal to that of the original liquid, would produce an osmotic pressure different from that of the cerebro-spinal fluid itself, and would therefore alter the freezing point.

The largest amounts of proteid were found in ten cases of general paralysis, a case of alcoholic delirum with polyneuritis, another of alcoholic senile deterioration with complete motor paralysis of the left side nine days before death, and in a case of organic dementia in which there was a tumor mass in the corpus callosum. In other words, in those conditions associated with gross organic lesions in which there was extensive destruction of the brain tissue, the proteid was in the greatest abundance. This fact was shown in my previous paper, although at that time no exact quantitative determinations were made. In the other cases the proteid content was usually low. In two cases (15 and 19) the na-

ture of the proteid material was determined. Both albumose and peptone were absent, the entire content being in the form of nucleo-proteid, and serum albumin and globulin. These nucleo-proteids are only found in organic pathological conditions; other cases yield merely the ordinary albumins of blood serum.

Cholin was tested for in all the cases, and in twenty-one of these it was quantitatively determined. The figures are given in terms of the platinum salt, from which the absolute amount of the alkaloid may be calculated, if one remembers that the combination contains 31.64% platinum. It was found absent in only four cases, alcoholic depressive hallucinosis, a senile paranoiac condition, melancholia and senile dementia. In general paralysis it was invariably present; also in one case of central neuritis, and two alcoholic cases with polyneuritis, one with delirium and one with the typical picture of Korssakow's disease. In a case of senile dementia, with clinical symptoms of emaciation, twitchings and rigidity, it was present, although unfortunately there was no autopsy to determine any axonal change in the Betz cells or the presence of a central Marchi reaction. In another case of senile dementia it was absent; in a third with marked confusion and delirium a small amount was detected. One case of senile depressive paranoiac condition with a termination in a typical central neuritis showed the absence of cholin; in another case of senile dementia it was found to be present. This latter is of some interest, because the clinical symptoms of the case showed an incompatibility with any products of myelin degeneration, but when the brain was sectioned some time later, a tumor in the splenium of the corpus callosum was found. A case of traumatic organic dementia with a tumor of the corpus callosum, yielded large amounts of cholin. In one case where it was absent in the fluid, the brain and cord were also negative. In four cases, it was found in the brain, cord, spinal ganglia and peripheral nerves, coincident with its presence in the cerebro-spinal fluid. In case 10, one of alcoholic delirium with a very rapid polyneuritis of the ascending type, cholin was found in all the peripheral nerves examined, and was parallel with the presence and extent of the Marchi reaction. It was also present in a case of melancholia with death from carcinoma of the uterus. Although on the whole the largest quantities of cholin were found in general paralysis, no parallel can be

established between the amount of that substance, the brain weight and the duration of the disease process. The highest figures, outside of general paralysis, were found in case 18 (0.780 grm.), but here the degeneration was in a recent active stage, an apoplectic seizure having taken place only nine days previous to death. Although the finding was not an absolute one, yet the largest amounts of cholin seemed to run parallel with the highest percentages of proteid, both being a measure of the extent of destruction in the central nervous system. Cholesterin was always absent, but, as in all secretions, phosphates and sulphates were found whenever tested for.

BIBLIOGRAPHY.

1. I. H. Coriat. A Contribution to the Chemistry of Nerve Degeneration In General Paralysis and other Mental Disorders. American Journal of Insanity, Vol. LIX, No. 3, 1903.

2. Waldemar Koch. Zur Kenntniss des Lecithins, Kephalins und Cerebrins aus Nervensubstanz. Ztschr. für physiol. Chemie, Bd. XXXVI, H. No. 2. u. 3.

3. W. D. Halliburton and F. W. Mott. The Coagulation Temperature of Cell-Globulin and its bearing on Hyperpyrexia. Archives of Neurology, Vol. II, 1903.

4. Julius Donath. The Rôle of Cholin in Epilepsy, 1902. Awarded the International Prize by the Craig Colony for Epileptics.

5. P. A. Levene. The Autolysis of Brain Tissue. Journal of Medical Research, October, 1903.

6. J. A. Sicard. Le Liquide Céphalo-Rachidien. Paris, 1902.

7. I. H. Coriat. Review of above. American Journal of Insanity, April, 1903.

8. S. J. Cole. On Changes in the Central Nervous System in the Neuritic Disorders of Chronic Alcoholism. Brain, Autumn, 1902.

9. J. Donath. Die Behandlung der progressiven Paralyse. Allg. Ztschr für Psychiatrie und psychisch-gerichtliche Medicin. Bd. LX, H. 4, 1903.

10. A Discussion on the Pathology of Nerve Degeneration. British Medical Journal, September 27, 1902.

11. F. H. Scott. On the Structure, Microchemistry and Development of Nerve Cells, with Special Reference to their Nuclein Compounds. Transactions Canadian Institutes, VI, 1898 and 1899.

12. S. Hatai. The Effect of Lecithin on the Growth of the White Rat. American Journal of Physiology, September, 1903. (This paper contains a complete bibliography.)

13. G. Marinesco. Recherches sur les Lésions des Centres Nerveux consécutives à l'Hyperthermie Expérimentale et à la Fièvre. Revue Neurologique, 1899.

14. Loc. cit.

15. E. Salkowski. Zur Kenntniss der Hydrocephalusflüssigkeit. Festschrift für M. Jaffé, 1901, S. 267-276.

16. I. H. Coriat. The Cerebro-Spinal Fluid in Hydrocephalus. American Journal of Physiology, November, 1903.

17. Torald Sollmann. The Chemistry of Cerebro-Spinal Fluids. Journal of the American Medical Association, June 6, 1903.

18. Maurice Dircksen. Etude sur la Composition Chimique et la Concentration Moléculaire du Liquide Céphalo-Rachidien. Thèse de Paris, 1901.

19. St. Clair Thompson. The Cerebro-Spinal Fluid, its Spontaneous Escape from the Nose, with Observations on its Position and Function in the Human Subject. 1899.

20. F. W. Mott. Note upon the Cholin Test for Active Degeneration of the Nervous System. Archives of Neurology, Vol. II, 1903.

In the Revue Neurologique for March 15, 1903, there is a fairly complete bibliography of the Cerebro-Spinal Fluid, but relating mostly to its cytology and cyto-diagnosis. In the same journal for March 31, 1903, there are a large number of papers on the same subject, being the Transactions of the Neurological Society of Paris.

REPORT OF A SECOND CASE OF DEMENTIA PRÆCOX WITH AUTOPSY.

By WILLIAM RUSH DUNTON, JR., M. D.,

Assistant Physician, Sheppard and Enoch Pratt Hospital, Towson, Md.

(*From the Laboratory of the Sheppard and Enoch Pratt Hospital.*)

In the AMERICAN JOURNAL OF INSANITY for January, 1903, I reported a case of dementia præcox with the findings after autopsy. Shortly after the work was concluded we were so fortunate as to have an autopsy on a very similar case and I was able to make a more thorough study of the general organs than had been done in the first case. The present paper reports the findings of the second case, an abstract of whose history follows:

Woman aged 37, single, no occupation, a native of Virginia, was admitted to the Sheppard and Enoch Pratt Hospital August 11, 1896.

F. H.—Both parents died of tuberculosis at 47 and 42 years, the patient being aged seven. Patient has four sisters living and well. A brother died in infancy.

P. H.—The only diseases which the patient had had were measles and whooping-cough when a child, and an attack of stomatitis, associated with some gastric disturbance, at twenty years. The patient went to school at five years, was attentive to her studies and learned readily, having no favorite topics. In disposition she was shy, retiring and seclusive. Later she led a very unselfish life, was busy at all times, and was devoted to her nieces and nephews with whom she was a great favorite.

P. I.—Four years before her admission, the patient went to live with a married sister whose husband was unkind to his wife and children as well as to others with whom he came in contact. It is probable that the patient suffered a good deal of mental distress from this cause.

Some months before admission the patient went to live in

the country with a favorite cousin who expected to be confined. One day in June the patient went to town and at noon telephoned to this cousin, learned that she was all right and arranged to spend the remainder of the afternoon in town. A few hours after, a person suddenly entered the room where the patient was and abruptly announced that her cousin was dead. For an hour afterwards the patient was completely prostrated. She felt it her duty to assume the care of her cousin's children and returned to the country to live. Her cousin's grave was situated in plain sight of the house and was a constant stimulus to depressing thoughts. The servants in the household used among themselves a language unknown to the patient and she soon began to think they were making her an object of ridicule. It is interesting to consider how in this case there was probably a gradual progress of her ideas, such as has been well described by Wernicke in his ideas of reference. At first the patient probably recognized that the ideas which were suggested by her brother-in-law's misconduct were largely the result of her own imagination, then as these continued and suspiciousness increased there came a time when she almost believed in them and finally they became well established delusions.

About a month before her admission she was found to be run down in health, supposedly suffering from malaria, and was sent to a sister who was staying in the mountains. This sister at once noted mental disturbance. The patient stated that on the train some one had been watching her. She soon showed an aversion, or even fear, of negroes with whom she had been associated all her life and tried to avoid meeting them on the street. She confessed to masturbation and was markedly self-accusatory. Later she apparently had hallucinations of sight and hearing, and insomnia and loss of appetite became marked. For a month before admission she complained almost constantly of headache.

On admission the patient was confused and restless. Her attention was difficult to obtain or hold. Her answers to questions were somewhat irrelevant. Her ideas were very incoherent. Leucorrhœa was found with tenderness of the genitals (uterus). Weight was 113 pounds. The patient took but little nourishment and had to be fed by a nurse.

She soon developed various delusions, hallucinations of hearing

and mixed identities of those about her; for example, she thought she was hypnotized by another patient whom she mistook for some one else she knew. Spoke of hearing rapping in her room at night. She was often apt to be emotional. For about a month after admission the patient's confusion deepened. She later became gradually less confused but hallucinations of hearing were very marked and controlled her actions. She frequently said that she must go away and showed marked motor restlessness. The patient showed circulatory disturbance, the pulse being soft and weak, and hands cyanotic. On one occasion when an attempt was made to take a sphygmographic tracing, she made active resistance and could not be made to understand the purpose of the procedure. Her skin was usually moist. In January, 1897, she is noted as being quite cheerful. A month later she was depressed and had a spell of crying about once daily. This condition lasting for a month or more. In July she began to answer the voices more than formerly, was restless and emotional. Impulsiveness was also noted at this time. She would occasionally soil herself while standing up. She rarely sat down. She still had to be fed by nurse. The night of August 28th, she was heard sobbing in great distress crying, "Oh, God! why am I tormented so?" On being questioned about it the next morning, the patient said she wished a certain chair removed from her room as it distressed her, giving as a reason that it was heavy, but said she had not lifted it. "It is heavy because of the iron in it, and it makes the room whirl." Patient's sister said she had frequently spoken of carriages, furniture, etc., being heavy but apparently did not mean weight by it. Her skin was usually greasy, her face flushed, and her hair in disorder. Dementia gradually became more marked and in January, 1900, it is noted that it is almost impossible to get a relevant answer to a question. She was still given to sudden impulses such as overturning furniture, swearing, etc.; these having gradually become more marked, and seemed to be the result of her delusions and auditory hallucinations. Restlessness was very marked, the patient asking to go out, to go into another ward, etc., and was very impatient until her request was granted when she would change and wish to do something else (negativism).

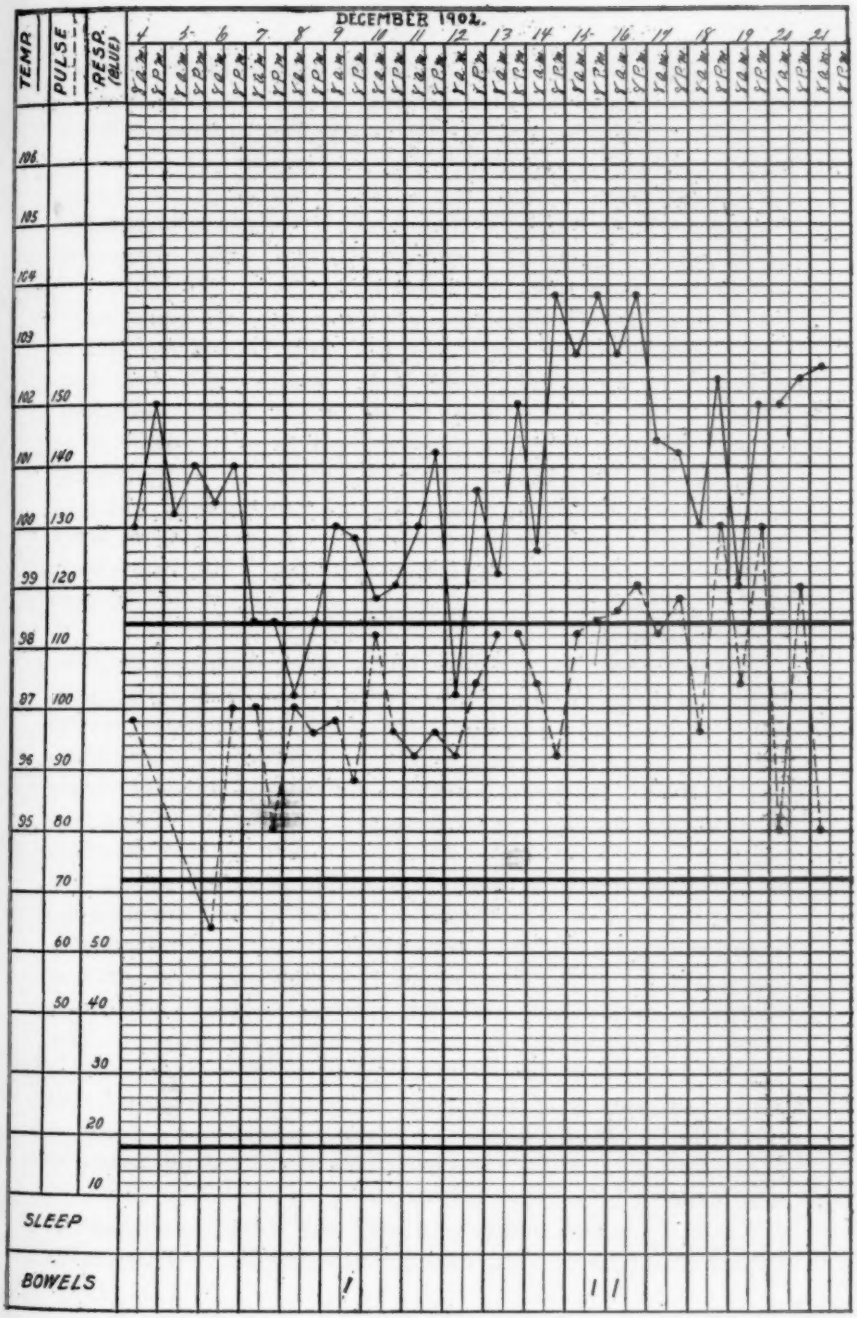
In January, 1901, it is noted that these sudden impulses led her

to strike nurses and patients but that recently she had been quieter and would occasionally play a game of cards or do some embroidery. She gained very much in flesh, her weight being 186½ pounds.

The patient showed stereotypy, preferring to sit in the same chair in the same place, and if it was occupied, frequently asking for it after a period of increased restlessness. She constantly complained of being tired, and often gave this as a reason for doing or not doing things. She often asked to have furniture, pictures, etc., moved and became very angry when it was not done. No systemization of her delusions could ever be positively made out, but on several occasions the patient referred to her husband, would often discharge the nurses when they refused to do her bidding, and in various other ways, led us to believe that there was some systemization of ideas. There seemed to be but little doubt that the patient was disoriented. Her sentences were usually left incomplete. In the letters which she wrote the writing was very irregular and unformed, but the spelling and phrasing better than would naturally be expected from her.

In September, 1902, the patient had a superficial thrombosis of the left leg and was put to bed, where she spent the greater part of the time until November, when she was gotten up for a short time each day. Early in December she began to spend more time in bed requesting to be allowed to do so. She usually lay in a very uncomfortable position, lying on her left side with her shoulder and head against the headboard of the bed. From March, 1901, the patient had gradually lost flesh, until she now weighed half of what she formerly did, her weight on December 8, 1902, being 93 pounds. Examination of patient's chest showed puerile breathing with slightly prolonged expiration on the right side. Examination of urine was negative. Chest examination made December 15, showed moist râles at the apex. Temperature is shown in annexed chart. Patient took very little nourishment. The following notes are copied from the history:

December 17.—Is weak and semi-delirious. Refuses food and medicine. Will take a tablespoonful of liquid nourishment at a time, but will not take sufficient in the twenty-four hours to properly nourish her. She breathes through her mouth constantly and resists efforts made to moisten it. Muscular movements


DECEMBER 1902.
TEMP
PULSE
RESP. (BLUE)
4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
A.M. P.M.
106
105
104
103
102 150
101 140
100 130
99 120
98 110
97 100
96 90
95 80
70
60 50
50 40
30
20
10
SLEEP
BOWELS

have been marked and during the recent high temperature a constant coarse tremor has been present.

December 19.—Pulse is weaker to-day but patient has lower temperature and seems better. She has emaciated markedly in the last few days.

December 20.—Patient's temperature is somewhat higher and she is rather weaker. She continues to have clonic movements and at times these become quite severe.

December 21.—During the night the patient was quite noisy and became unconscious. About 5 A. M., she was not able to swallow and would hold liquid in her mouth for a long time. About 2 P. M., her pulse was 156, respiration 48, Cheyne-Stokes, her extremities were cold and the movements of her hands were quite marked. She apparently was conscious as she moved her eyes toward speaker and apparently tried to respond. At 3.45 her respiration had become regular and she was trying to articulate and in doing so almost had a convulsion. Her hands were markedly flexed and thumbs turned in. Patient died at 6.30 P. M. quite quietly.

To recapitulate, the patient exhibited the following symptoms: In the beginning she showed depression, delusions of persecution, self-accusation, probably visual and aural hallucinations, mental confusion, motor restlessness, mixing of identities, weak attention; and physically, headache, insomnia, and loss of appetite. Later the confusion and depression decreased, there were frequent outbursts of excitement due to the hallucinations of hearing which became more active, impulsiveness was present, the patient was untidy, she showed disturbances of circulation, and dementia became gradually more and more marked. The usual accompanying symptoms of this later stage of dementia præcox, such as stereotypy, negativism, irrelevant answers (Gänser's symptom) were present and impulsiveness became more marked. Her weight throughout showed a characteristic curve (see chart). Finally the patient became more resistive, more demented, lost rapidly in weight and died.

The autopsy was made one hour after death and the following was noted:

The body is very much emaciated and subcutaneous fat has largely disappeared. The mammæ are atrophied. There is no

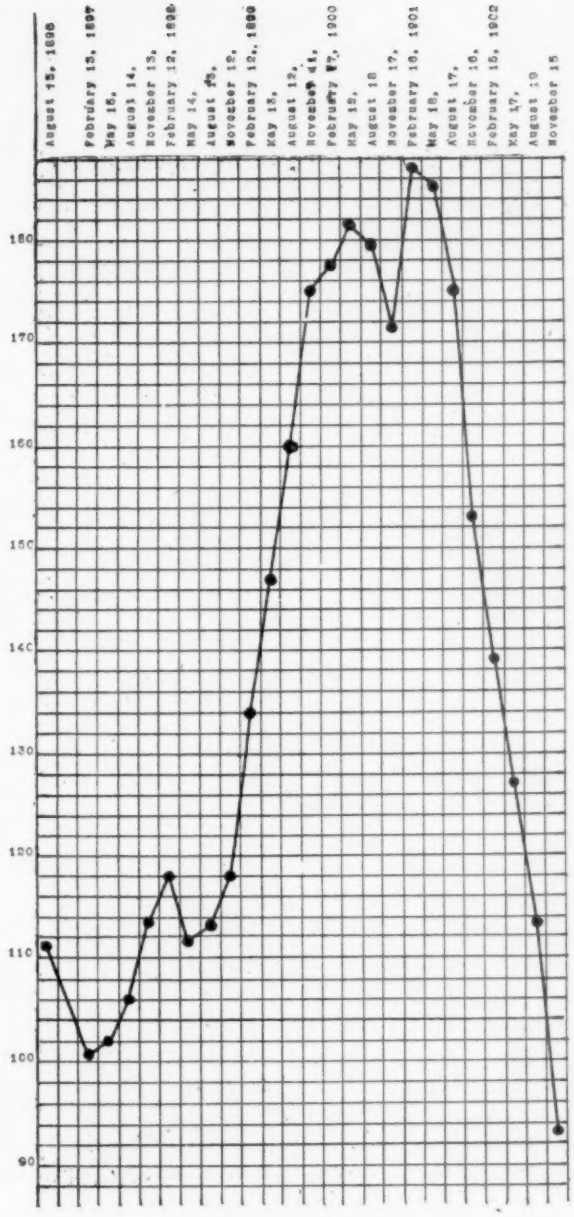


CHART SHOWING WEIGHT.

post-mortem rigidity. Some discoloration of the dependent parts is present. A bed-sore about an inch and a half in diameter is seen over the sacrum to the right of the spinal column, and a smaller one over the right external malleolus.

On removing the skull-cap the dura is found to be more adherent than usual over the region of the first frontal convolutions to within an inch of the coronal suture. It cannot be separated from the skull without tearing. A moderate amount of cerebro-spinal fluid is present. The brain is symmetrical and the convolutions are somewhat atrophied. There is a general engorgement of the vessels and apparently some increase in their number. Lying over the hemispheres there is a velamentous layer which folds back with the dura and looks like fibrin. This has no organic connection with either pia or arachnoid.[1] There are no adhesions between the dura and the pia. The vessels at base of brain and the larger branches extending up over it appear to be normal.

The spinal canal seems to contain an excess of fluid. There are numerous small areas of calcification about 2-4 mm. in diameter in the arachnoid. There is no cloudiness of the fluid.

The abdomen is slightly distended and the cavity is dry. The omental fat is very scanty. The colon is distended. The color appears normal. The appendix is 6 cm. long, has a mesentery for half its length and extends over the brim of the pelvis. There is a concretion about 1 cm. in its extremity. The small intestine is intensely engorged for several feeet. The lymphatic system is quite prominent, being apparently dilated.

Lungs.—There are adhesions between the apex and upper half of the right lung and the thoracic wall. The upper and middle lobes are hard, the lower collapsed. On section the upper lobe shows gray hepatization with an area of broken-down tissue about 2 x 5 cm., lying at the apex near the surface. The middle lobe is congested and shows numerous white areas from 1 to 5 cm. in diameter. These are not raised.

The left lung has several adhesions at the apex and is collapsed.

Thyreoid.—Normal except slight asymmetry of the lobes.

[1] Kiernan, in AMERICAN JOURNAL OF INSANITY, Vol. XXXIV, p. 59, 1877, notes a mucoid material found as an arachnoid exudate. This he considers to be a "simple infiltration of molecular matter and blood-discs," rather than an inflammatory product.

Trachea.—Normal.

Aorta.—Normal.

Œsophagus—Thoracic Duct.—Normal.

Heart.—The pericardial cavity contains a very small amount of fluid. The heart is small. The valves are competent. The heart-muscle is slightly pale. The cavities contain red post-mortem clots. The endocardium is clear. There are a few patches of sclerosis about the coronary vessels, which latter are normal.

Liver.—The liver feels slightly harder than normal. It weighs 950 gm. On section it shows light spots 1 to 3 mm. in diameter, not raised but palpable. The gall-duct is clear. There is one gall stone in the gall bladder.

Kidneys.—The right kidney measures 10.5 x 5 x 3 cm., and weighs 115 gm. The left measures 11.5 x 5 x 3 cm., and weighs 125 gm. The capsule of the right is slightly adherent and some of the parenchyma strips off with the capsule. Its color is somewhat darker than normal. The stellate veins are unusually prominent. On section it cuts firmly with a gritty sensation. The cortex measures 6 mm. in thickness. The pelvis of the kidney is negative. The vessels of the cortical substance are fairly distinct. The Malpighian bodies are just visible.

The left kidney is similar in appearance.

Suprarenals.—The right suprarenal was torn in taking it out. It is cystic in the medullary portion. The left measures 6.8 x 2. 2 x 8 cm., and weighs 10 gm. It is normal in appearance.

Spleen.—The spleen measures 10 x 7.5 x 3.2 cm. and weighs 115 gm. On section it is very dark, and softer than normal. There is no increase of connective tissue in it.

Pancreas.—The pancreas is small and weighs 60 gm. It appears normal on section.

Ovaries.—The left ovary is small, wrinkled and attached to or enclosing a cyst about 3 cm. in diameter filled with a dark brown, thick fluid. The right ovary is smaller, more wrinkled and spongy. It is in intimate connection with a smaller cyst filled with a fluid similar to that noted above.

Uterus.—The uterus is small and hard. The external os is extremely hard.

Anatomical Diagnosis.—Chronic atrophy of brain; fibrinous

exudate; gray hepatization of right lung; atelectasis of left lung; cirrhosis of liver; slight splenic tumor; hemorrhagic infarct of intestine; double ovarian cyst; right cystic adrenal.

Microscopical examinations of the various organs were made after hardening according to Zenker's method and in bichlorid solution, and staining with hæmatoxylon and eosin and Mallory's connective-tissue stain. Briefly the results of these examinations are as follows:

Lungs.—The right shows fibrinous pneumonia, broncho-pneumonia, and gray hepatization. The left shows the same changes to a less degree. Sections stained for tubercle bacilli were negative.

Kidneys.—The right shows an acute diffuse nephritis, the left an acute diffuse hemorrhagic nephritis.

Suprarenals.—Both right and left were examined. The cortex of the left shows small areas of fatty degeneration.

Heart Muscle.—Negative.

Voluntary Muscle.—A portion of the left sartorius was taken for examination and is negative.

Spleen.—Shows acute splenic tumor.

Pancreas.—Shows parenchymatous degeneration.

Appendix.—Shows some fibrinous exudate but is otherwise negative.

Stomach.—Shows a condition of chronic gastritis.

Liver.—shows an atrophic cirrhosis with fatty degeneration.

Thyreoid.—Both lobes are negative.

Pineal Gland.—Negative.

Dura.—Capillary hemorrhages are occasionally seen. Several concentric bodies, or psammoma are also seen surrounded by round cells. Sections of dura stained by Kuhne's, Gabbet's and Gram's methods for bacteria were negative.

Pia-Arachnoid.—The pial vessels are engorged, there is a slight proliferation of the endothelial cells upon the surface, and about some of the larger arachnoid spaces. There are quite a number of recent capillary hemorrhages on the surface of the cortex. The velamentous layer of fibrin referred to in the autopsy notes was stained by hæmatoxylon and eosin and showed layers and masses of fibrin among which are numerous leucocytes. Sections stained with gentian violet and by Gram's method to show bacteria, are negative.

Peripheral Nerve.—The external cutaneous nerve is negative.
Choroid.—Negative.
Basilar Artery.—Negative.
Aorta.—Negative.
Sympathetic Nerve.—Negative.
Small Intestine.—The injected portion shows the glands swollen and congested. There are numerous minute areas of recent hemorrhage in the mucosa, some, however, being quite extensive, and the whole mucosa is pretty generally infiltrated with red blood-cells. Leucocytes are generally distributed through these areas. There are a few areas of small round-cell infiltration.

The findings are those which are usually found in cases dying of starvation. It must be remembered that the patient had been losing weight for several months and during the last three weeks of her illness it had been impossible to get her to take but a minute amount of nourishment so that the cause of her death was really starvation. No other changes were found except in the nervous tissue which indicated that pathological changes were present before those caused by the changes due to the starvation.

For a study of the nerve-cells, tissue was taken from the right and left paracentral convolutions, the right and left first frontals, left anterior and posterior centrals, left temporal, occipito-calcarine, cuneus, Broca's, superior angular, left thalamus, the cerebellum, cord, third nerve, and inferior portion of the Island of Reil. These tissues were fixed in alcohol, and nitric acid, and subsequently stained by the Nissl and Bethe methods, and Unna's polychrome methylene-blue stain.

In the study of the case of dementia præcox which has already been reported the nerve-cell findings were reported in detail, but in the present study this seems unnecessary and the following summary indicates the findings: Pale yellow pigment is seen in increased amount in the majority of cells, central chromolysis is frequent. The cell is often swollen. The nuclei are atrophied, swollen, displaced and distorted. An endonucleolus is occasionally present. Folding of the nuclear membrane is rare. Neurophagocytosis is occasionally seen. The glia nuclei are slightly increased about the cells, and occasionally cover them. Fragmentation of the cells is rare. The small pyramidal cells are frequently seen twisted upon their axes.

The cerebellum showed a decrease in the number of Purkinje cells, and those present were distorted, atrophied, and showed the condition known as "ghost cells." The nuclei showed atrophy and distortion. There were but few cells which appeared normal. In all cells the chromoplasm was much broken up and the cells presented a granular appearance. There was no notable increase of neuroglia or of the cells described by Lannois and Paviot. (See note 4).

Comparing these findings with those of the case previously reported we find that there is an increased amount of pigment observed, atrophy is not seen quite so often, nor is folding of the nuclear membrane. Cell disintegration is less frequently observed and the presence of an endo-nucleolus is not so constant. On the whole I would consider that the changes observed in this case indicate that the pathological processes are in a less advanced degree than in the first case. In this connection it is of interest to recall several facts—first, this case did not die of tuberculosis as did the former; second, that the degree of dementia was greater in the former case than in the present case; third, that the date of onset and date of death occurred later in the life of the individual; fourth, that the course of the disease was more prolonged. It seems as though these differences would probably account for the slight variations in the cell findings of these two cases.

Sections stained by the Bethe method showed the intra-cellular fibers distinctly stained except in the central part of the cell where chromolysis was supposedly present, and here there was a granular appearance. In some cases this involved nearly the whole cell. With this stain the pale yellow pigment was more marked than with the Nissl staining.

Sections were stained by the Weigert-Pal method, and from a study of them there seemed to be no doubt that the tangential fibers were decreased in number.

For the study of the neuroglia, sections were stained by the Benda and Weigert's methods. Unfortunately neither of these methods was entirely successful, so that I am unable to state whether there was an increase or decrease of neuroglia fibers. There was undoubtedly an increase of the neuroglia nuclei as could also be seen from sections stained by the Nissl and polychrome stains. This increase was more marked about the cells

and was only slightly present about the vessels. Sections stained with eosin and hæmatoxylon showed no pathological changes in the vessels of the cortex, excepting that in the cuneus there was observed a small round-cell infiltration about some of the vessels.

In sections stained by Weigert's mitosis stain, after careful study, I was unable to find any mitotic figures in the glia cells.

There were several unusual phenomena which occurred while the tissue of this case was being stained which deserve mention in some detail, but I am of the opinion that until these phenomena are explained, we should not attach too much importance to them. In staining certain portions of the cerebrum fixed in 96 per cent alcohol with the polychrome stain, the white matter retained the color longer than the cortex, as is seen when an attempt is made to stain chromicised tissue with methylene blue, but on further decolorization this portion of the section lost its color. It seems to indicate some unusual chemical change in the brain. It was seen most markedly in tissue from the paracentral convolution, but also in tissue from the first frontal. Further, a number of sections of formalin fixed tissue from the frontal region, stained with hæmatoxylon and eosin, showed blue lines in the cortex which with low power suggested glia fibers, but which when viewed with oil immersion were found to be broad, somewhat irregular and with swellings. These fibers were most abundant a short distance outside of the medullated layer, and the larger ones lay perpendicularly to the surface but were crossed by a number of finer one which lay irregularly or parallel to the cerebral surface. Their appearance and the manner of their disposition suggested myelin sheaths. Frequently two fibers would lie closely parallel. The larger ones were frequently seen lying in a space. This same tissue was stained most successfully by the Weigert-Pal method and there can be no doubt that owing to some unknown chemical change the myelinated fibers reacted to the methylene blue and the hæmatoxylon stain. It is the first time that such an unusual circumstance has been observed in our laboratory.

In other sections stained by hæmatoxylon and eosin there were seen in the white matter a number of small areas stained bluer than the surrounding tissue. While these phenomena may have been due to some accidents of technique all the steps have been carried out most carefully and this explanation does not satisfy

me, and I am inclined to think that there was some unusual chemical change in the cortex which caused these reactions.

Two writers have recently investigated the pathological anatomy of dementia and it is of interest to compare them with those in the writer's two cases. Lubouchine,[2] after study of two cases of dementia præcox found that the nerve-cells were atrophied and showed fatty yellow degeneration, that there was an enlargement of the perivascular spaces, a proliferation of neuroglia in the molecular layer and in the layer of the small pyramidal cells, and about the blood-vessels. There was also degeneration of the subcortical or tangential fibers. He believes that this diffuse atrophic and degenerative process takes a slow course. The other writer, Bridier,[3] finds that the cortical lesions involve the nerve-cells, the supporting tissue, and the vessels. In the cells there is fatty and pigmentary degeneration, central chromolysis, division of the nucleus, and finally more or less slowly, atrophy of the nerve-cell. Proliferation of neuroglia invades the layers of pyramidal cells, migratory elements appear, and there is arterio-sclerosis of the vessels. He also found changes in the cerebellum which resemble those described by Lannois and Paviot[4] that is atrophy and rarefaction of the Purkinje cells and the presence of a granular layer situated below them.

It has been noted by some writers that the number of nerve-cells in the cortex is diminished in cases of dementia. On this point I am of the opinion that our knowledge of the normal distribution of nerve-cells in the human cortex is not yet exact enough to allow us to make or to accept such positive statements, excepting possibly in senile cases on idiocy. A number of writers have made reports of their investigation of the distribution of

[2] Modifications anatomo-pathologiques de l'Ecorce Cerebrale dans deux cas de Demence precoce. Journal de Neuropathologie et de Psychiatrie du nom S. S. Korsakoff, 1902, livre 1-2, p. 61-72. Abstract in Revue Neurologique, An. XI, p. 69, 15 Mai, 1903.

[3] Essai sue l'Anatomie Pathologique des Demences. Thèse de Lyon, 1902. Abstract in Rev. Neurologique An. XI, p. 620, 30 June, 1903.

[4] Les Lesions histologiques de l'ecorce dans les atrophies du cervelet. Nouv. Icon. de la Salpêtrière, An. 15, No. 6, p. 513, November and December, 1902.

nerve-cells in different areas of the cortex and in time the whole cerebrum will be fully studied. I have under way such an investigation of the first frontal convolutions which I feel sure will prove of value both from an anatomical and pathological standpoint, but at present I do not feel competent to express a positive opinion as to the nerve-cell distribution in this case. From the accompanying photograph (Fig. 1.) of the apex of the left anterior central convolution it will be seen that at least there is no marked disappearance of the cortical nerve-cells.

The changes found in the nervous tissue in this case, and that reported previously, confirm in general those observed by these other writers. The changes observed in other organs are negative when taken in connection with the mental disease.

The uniformity of the findings would seem to indicate that the pathology of dementia præcox may be considered as becoming established, though as yet we must keep the question open until other reports further confirm these findings. That similar findings have been reported in animals dying of experimental toxæmia is simply confirmatory of theories which have been advanced in the past by Nolan, von Tsisch, Bernstein, and others, that the condition is caused by an autointoxication probably emanating from the reproductive organs. The recent work of Bruce and Peebles,[6] in which they claim to have isolated a specific streptococcus needs confirmation, as these writers themselves suggest.

In my first case, I was of the opinion that the tuberculosis, from which the patient suffered, had not had any especial influence upon the pathological findings. In the present case I am of the opinion that the malnutrition, or starvation from which the patient suffered had no especial influence upon the changes in the nervous tissue.

From a study of the pathological changes as well as from the clinical histories, the conclusion seems justifiable that in dementia præcox we are dealing with a degenerative psychosis, probably of autotoxic origin, and of slow course.

[6] Journal of Mental Science, Vol. XLIX, p. 614.

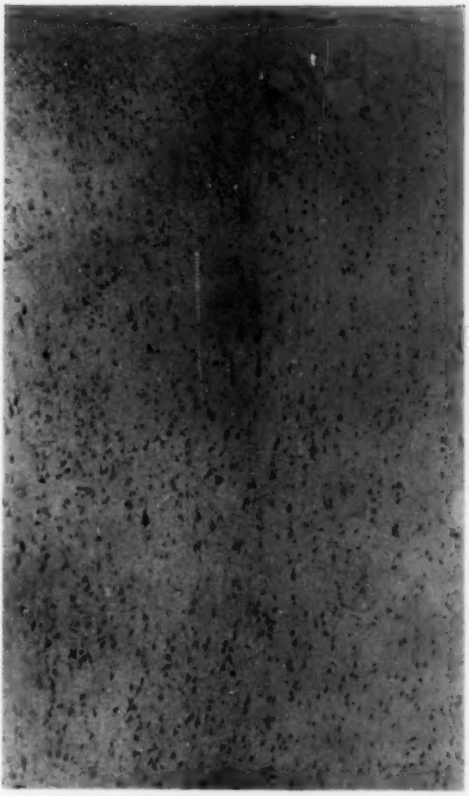


Fig. 1.—From anterior frontal region showing arrangement of cells.

A CASE OF CHOREA INSANIENS WITH REPORT OF AUTOPSY.[1]

By ISABEL A. BRADLEY, M. D.,

Pathologist and Assistant Physician, Columbus State Hospital.

The case which I will present to you in this paper is one of unusual interest, not only because of the rarity of this mental state, but because it presents several pathological conditions which have been subjects of investigation for some time. Keenly realizing that the work is incomplete, I still believe that it is not entirely without value.

Mrs. E. R., age 19, a native of Ohio, was admitted to the Columbus State Hospital, Nov. 11, 1902, and died at the Hospital Nov. 13, 1902, forty-three hours after admission. The medical certificate of her commitment papers gives the following evidences of insanity, " outbreaks of excitement, during which she is uncontrollable, delusions of persecution, unduly suspicious and at times incoherent."

The patient had not been well for about two weeks before coming to the hospital but had been cared for at her home. The only history obtainable concerning her parentage was that her mother was a very nervous and peculiar woman and was separated from her husband. There was no history of insanity or epilepsy in the family. The patient had always been nervous. This nervousness had increased since puberty. She was of a kindly disposition and always industrious. No history of previous sickness could be obtained. During the summer previous to her death she had nursed her mother through a long siege of typhoid fever. Seven weeks before her admission to the hospital she was married. She frequently complained of being tired, and gradually became more nervous and restless and slept very poorly. Several

[1] Read before the meeting of the Association of the Assistant Physicians of the Ohio State Hospitals, held in Toledo, Ohio, October 7-8, 1903.

days before coming to the hospital she developed irregular, jerky movements of the body which were greatly exaggerated at times. At this time she had some control of herself, although occasionally this would be entirely lost. She also had convulsive seizures which were considered by her attending physician to be of the nature of hysteria major. She grew very suspicious of people and took a great dislike to doctors.

She was brought to the hospital about 4 P. M. Nov. 11, 1902. Her hair was down her back, her shoes off and she was only partially dressed. The movements of her body were such that she could neither sit nor stand. She would thrust first one hand and then the other through her hair. The trunk would bend to one side, then to the other and at times the whole body would give a twist. She would toss her head and jerk one limb and then the other.

She was conscious and orientation was normal. She knew that her friends thought her insane.

Shortly after admission she took some bread and milk, and that evening went to the dining-room and ate some supper. She objected to going to bed and having a tray brought to her. Physical examination was impossible on account of the movements of the body. She appeared anæmic and had a rapid pulse. During the night she remained in bed fairly well but was restless and tossed about considerably, sleeping only a few moments at a time. The following day she was more restless and wanted to go home. She was quite incoherent and irritable. She took nourishment only when urged to do so. She was taken out-of-doors during the day, as she appeared much quieter when out and was very anxious to go. That night when put to bed she began to toss about and very rapidly became worse. She would throw herself about, would sit up, then get on her knees or toss her head. She would throw her arms and legs about in such a way as to bruise them. She paid no attention to questioning and had absolutely no control of herself.

At 7.45 P. M. that evening she was put in a wet-sheet pack with cold cloths to her head. This did not quiet her movements. She continued to throw her head about and bite her tongue. At 9 o'clock she was taken out of the pack and restrained in bed. One-fiftieth of a grain of hyoscine hydrobromate was given

hypodermically. The movements became more violent. She would scream, roll her eyes, protrude her tongue and quickly jerk it back, close her jaws firmly, blow through her nose forcibly, throw her limbs about and raise the trunk. These paroxysms would last from ten to fifteen seconds and would then be followed by hard breathing. Unless restrained the patient would injure her limbs by striking the edge of the bed. She was placed in a wet sheet wrung out of warm water and cold cloths were kept on the back of the neck and forehead. The face became flushed and for a time the changing of the cloths seemed to start the paroxysms. A small feeding tube was passed into the stomach and a pint of milk and a pint of water were introduced. Fifteen grains of chloral hydrate and thirty grains of bromide were also given.

At 1.00 A. M. the motor symptoms still persisted, but the crying-out had ceased. About 3 A. M. the motor symptoms subsided and the patient became comatose; the breathing was stertorous in character, the pulse was running and the temperature rising. At 6.30 A. M. the temperature was 103.1° per axilla, the pulse running and respiration irregular. At 8 A. M. the respirations were markedly Cheyne-Stokes. The patient died at 11 A. M.

I am indebted to Dr. G. T. Harding, Jr., the attending physician, for the clinical notes.

The *autopsy* was commenced two and one-half hours after death. The corpse was that of a young woman of small stature, well formed and with regular features. The nutrition was poor. The panniculus adiposus was very small in amount, the musculature small. Rigor mortis was appearing in the jaws. There was some hypostasis in the dependent parts. The eyes were prominent; the pupils widely dilated and equal. There were several abrasions on the lips where they had been bitten. The skin over the abdomen showed numerous superficial linear abrasions caused by the movements of her hands when in the pack. There were numerous black and blue spots on the leg. The fists were clenched and the feet extended.

Head.—The scalp was normal. The skull was thin. The dura was of normal thickness and not adherent to the calvarium. The longitudinal sinus was empty. The tension of the dura was markedly increased. The under surface was smooth and glisten-

ing and was not adherent to the pia. The vessels of the pia were brightly injected. This was most marked in the posterior portion of the superior parietal regions. There was a considerable amount of clear, yellow fluid under the pia. The latter stripped from the convolutions easily. The consistence of the brain substance was considerably diminished. This was not so marked in the occipital regions. On section the gray matter had a glistening, swollen appearance, the natural divisions not being clearly defined. The blood-vessels throughout the brain substance were seen as bright red pin-points. The blood-vessels at the base of the brain were normal. The cerebro-spinal fluid was increased.

Thorax.—After the sternum was removed, in the upper part of the anterior mediastinum was a small pinkish body which in location and general contour corresponded to the thymus gland. It presented two well-defined lobes, the right lobe being about 2 cm. longer that the left; it weighed a little over 8.5 grams and measured 5 cm. in length, 4.5 cm. in width and .6 cm. in thickness.

The heart weighed 185 grams and appeared small for the size of the body. In the pericardial sac there was about 15 cc. of clear, yellow fluid. The left ventricle was firmly contracted. The heart was empty, except that in the auricles there was a small amount of fluid blood. The auricular surface of the mitral flaps was thickly studded with small roundish, white vegetations which were firmly adherent to the underlying tissue. The aorta, pulmonary and tri-cuspid valves were normal. The heart-muscle was normal in appearance.

The *left lung* weighed 160 grams and was alherent to the chest wall and diaphragm; the adhesions were easily torn. Otherwise the lung was normal.

The *right lung* weighed 250 grams and, aside from a small deeply-congested area in the posterior middle portion of the upper lobe, was normal in appearance. There were several small, caseous bronchial glands.

Abdomen.—The peritoneum had a dry, dull appearance. The position of the organs was normal. The spleen weighed 125 grams. The capsule was smooth and distended. On section the pulp was increased somewhat. The stroma was fairly distinct and the follicles slightly increased in prominence.

The kidneys weighed 120 grams each and but for deeply congested pyramids, appeared normal. The liver weighed 1190 grams. There was an increased amount of blood in the parenchyma.

The pancreas and adrenals appeared normal. The stomach contained about 250 cc. of a dark greenish fluid with a few semisolid particles having the odor of sour bile. The mucosa of both stomach and intestines was pale but otherwise normal.

The uterus was pregnant and contained a fœtus 4.5 cm. long and was probably about ten weeks old. The ovaries were of normal size. The right ovary contained a corpus luteum verum. The cortices of both ovaries contained Graaffian follicles in different stages of development. The bladder contained about 60 cc. of dark turbid urine. The mucosa was pale.

The mesenteric lymph-glands were markedly enlarged. The inguinal and axillary glands were of normal size. The hemolymph glands presented a pinkish color, and one, a bright red color.

The aorta was small, measuring only 3.25 cm. in circumference in the abdominal portion. The intima was smooth and glistening, with no areas of degeneration. The elasticity was normal.

Anatomical diagnosis.—Œdema of the brain, pregnant uterus, persistent thymus and partial lymphatic hyperplasia, arterial hypoplasia, organized mitral vegetations, congestion of the liver, spleen and kidneys; caseous bronchial glands.

Microscopic examination.—The kidneys and liver showed an active congestion but were otherwise normal. In the spleen there was an endothelial hyperplasia throughout, which was most marked in the centers of the Malpighian follicles. The center of some of the follicles was becoming hyaline.

The mesenteric lymph-glands showed an endothelial hyperplasia. The hemorrhagic area in the lung had a thin connective-tissue wall which separated it from the surrounding lung tissue. The heart-muscle, pancreas and adrenals were normal.

The appearance of the thymus was that of an actively functioning organ. The adenoid tissue was abundant and made up of dense masses of lymphoid cells with very deeply-staining nuclei. The characteristic bodies of thymic tissue were numerous. Only one Hassel's corpuscle was seen which showed calcareous de-

generation. There was some congestion of the blood-vessels in the interlobular connective tissue. The hemolymph gland examined had its sinus and blood-vessels distended with red blood-cells.

Cortex.—With the naked eye the sections of the cortex could be seen to contain many small, clear spaces, both in the white and gray matter of the cerebrum. In the cerebellum they were confined to the white matter. In the sections examined these appeared to be somewhat more numerous in the central and parietal regions than in the frontal convolutions. These clear spaces were for the most part round and the largest were fully 5 mm. in diameter. Under the microscope the largest spaces appeared to be independent of the blood-vessels, but the smaller openings were due to dilated perivascular and pericellular lymph-spaces.

A section from the lower portion of the left posterior central convolution.—Large pyramidal cells. The protoplasm was very faintly and diffusely stained but the outline of the cells was distinctly made out. Many cells contained vacuoles in the periphery of the protoplasm. Some cells were mere shadows. The processes were very distinctly visible for a long distance and were stained a diffuse blue.

The nuclear membrane was entirely absent in many cells, the nuclei being indistinguishable from the surrounding cell-body. The nucleoli were very deeply stained, of about the normal size, usually normally located and many contained light areas.

The outline of the smaller pyramidal cells of the second layer was in most cases not distinguishable. The cells appeared to be disappearing and all the evidences of degeneration were in a more advanced stage than in the largest pyramidals. The cells of the frontal convolutions were similar to those in the central convolutions except that the protoplasm was more faintly stained and absolutely homogeneous; the nucleoli being small and very densely stained and having the appearance of being nearer the eye than the surrounding protoplasm. This was due to the advanced stage of degeneration of the protoplasm. The changes in the occipital lobe were not so advanced; the outline of the cells was distinct; the nuclei were definable, although the membrane was not visible and the nucleoli did not contain light areas. There were no nuclear folds in any of the sections examined.

The neuroglia did not appear to be increased in amount, although no special neurogliar stains were used. Several neuroglia nuclei were very deeply stained and had a notched outline similar to those described by Hoch.[3]

Pieces were taken from the first and second frontal convolutions of both sides of the brain, left central convolution, right superior parietal, right occipital, lenticular nucleus and cerebellum and stained by Nissl's methylene-blue method and hematoxylin and eosin.

The spinal cord was not examined.

Summary of the examination of the cortex.—The changes in the cells were fairly constant, although more marked in the central, parietal, and frontal convolutions. The second and third layers of the cortex seemed to have suffered the most. The changes consisted in a diffuse chromatolysis of the entire cell, the protoplasm taking a very light blue and having a crumbly appearance. The processes were abnormally visible, due to the achromatic substance taking the stain. The nuclei were small and the nuclear membrane was entirely absent. The nucleoli contained light areas and were usually small. The smaller pyramidal cells of the second layer showed the most advanced changes. The perivascular and pericellular dilatations were most extensive in the parietal and central convolutions. The very large, clear spaces were not seen in the sections of the lenticular nucleus or the cerebellum, but in these localities there were numerous perivascular dilatations.

It is greatly regretted that no bacteriological examination was made in this case, nevertheless, since there was no evidence of a gas-producing organism being present in any other organ of the body it does not seem likely that the openings in the brain substance were due to such a cause. It is most probable that there was a genuine œdema of the brain. Although there was a hyperæmic condition of the meninges and brain substance, there was no inflammatory condition. The elevation of temperature might have been due to the œdematous condition of the corpus striatum, for there is considerable evidence that lesions in the corpus striatum and optic thalamus produce elevation of

[3] American Journal of Insanity, Vol. 54.

bodily temperature.[3] Mitchinson,[4] reports two cases of rapidly fatal maniacal chorea and in both cases there was œdema of the brain. Bertram Abrahams[5] describes the cortical changes in a fatal case of acute chorea. They were very similar to those found in this present instance. He says, "The perivascular and perineuronal lymph-spaces were much dilated and the cortex was distinctly œdematous." Dana,[6] describes a case of chorea in which the white matter of the brain was honey-combed with little spaces.

The points to be emphasized in the clinical history are a neurotic hereditary taint, a nervous temperament, physical exhaustion followed by the connubial state, delusions of persecutions, hysterical phenomena, choreic movements, incoherence, delirium and coma.

Summary of pathological findings.—Œdema of the brain with advanced degeneration of its most highly specialized elements, the ganglion cells; organized mitral vegetations, congested hemolymph glands, arterial hypoplasia, an apparently actively functioning persistent thymus and mesenteric lymphatic hyperplasia.

The mental symptoms developed before the chorea but were very soon followed by the hysterical phenomena and the choreic movements. That the attacks in which the patient screamed, ran her tongue in and out, forcibly blew her nose, rolled her eyes about and raised the trunk of her body from the bed, were different from the general and constant twitching and throwing of her limbs and body can not be denied. The former no doubt belonged to a hysteria which accompanied the chorea. The gravid uterus was a predisposing factor in the production of the chorea. Often those who become choreic as a complication of pregnancy have suffered from chorea or rheumatic fever in youth. The absence of rheumatism or other infectious diseases from the history and the presence of the organized vegetations on the flaps of the mitral valve is significant.

The presence in this case of the lymphatic constitution as described by Ohlmacher,[7] is interesting and suggestive. While

[3] Lancet, June 29, 1889, p. 1395.
[4] Lancet, May 11, 1889.
[5] Lancet, February 10, 1900.
[6] Brain, 1890.
[7] The Bulletin of the Ohio Hospital for Epileptics, Vol. I, Nos. 2 and 3.

the picture is not as pronounced as in most of his cases, yet the apparently actively functioning, persistent thymus and the hyperplasia of the mesenteric lymphatics with a small aorta is, I think, sufficient to consider this a case of constitutio lymphatica.

The lymphatic constitution may be an anatomical evidence of a neurotic constitution, but the cause of the hyperplasia of one set of organs and the hypoplasia of another set of organs is still unknown. That a toxic agent is concerned seems most probable and that the condition is an auto-intoxication due to a disordered metabolism is also very probable. I believe the same poison in the system which caused the endocardial lesions, the changes in the spleen and the congested internal organs, in the exhausted and highly susceptible organism, caused the œdema of the brain and the changes in the cortical cells and produced the terrific mental and motor phenomena which we saw fit to call maniacal chorea.

Our pathological studies will permit us to see the ravages of this poison which is circulating in the fluids of the body, but we must carry our investigations into the domain of chemistry before the real cause can be determined.

INTRACRANIAL PSAMMO-SARCOMA WITHOUT PARALYSIS.

By WALTER D. BERRY, M. D.,

Clinical Director and Pathologist, State Hospital for the Insane, Waterbury, Vt., Professor of Mental Diseases, University of Vermont Medical College.

In the consideration of intracranial growths, we naturally look for grave and serious trouble in consequence of their presence within the brain cavity. One very naturally supposes that any interference with the cortical neurons either from direct or indirect pressure causes a more or less profound set of symptoms. In other words we expect some interference with the stream of thought or certain defined or ill-defined signs of motor-paralysis. If we do not find such pathognomonic signs or "the classic symptoms of brain tumor" we are dumfounded if at the post-mortem table we discover a large neoplasm which has undoubtedly occupied a large part of the space intended for the motor area and caused direct pressure upon the basal ganglia. Such however was true in the case I am about to present.

Intracranial tumors among the insane are not as common as one would naturally suppose them to be. In one hundred and twenty autopsies performed in the Vermont State Hospital during the last three years only one distinct intracranial neoplasm has been found. We are told that the brain is often damaged by new growths which arise in its substance or spring from the enclosing membranes or from the bones of the skull. However frequent this may be, yet certain unique features of this individual case together with its comparative statistical rarity are my reasons for bringing the case to your attention.

There seems to be considerable difference of opinion however regarding the name and true nature of a psammoma, although most authorities regard them as belonging to the class known as sarcomata. For instance one author says "angio-lithic sarcoma"

another "acervuloma cerebri," and still another authority contends that it "represents no distinct species of tumor."

Senn says "A psammoma is an endothelial outgrowth of the envelopes of the brain that are first described by Virchow who included it with the sarcomata. Bland Sutton refers it to an epithelial matrix in the villous processes of the choroid plexus; but as it is found more frequently in localities where there are normally no epithelial cells, it is advisable to include it among the connective tissue type of tumors. The tumor is composed of onion-like cell-masses separated by a stroma of connective tissue. The concentric bodies consist of endothelium-like cell-nests arranged around blood-vessels which in the course of time become infiltrated with calcareous salts. It was first believed that the dura mater was the favorite seat of psammoma, but more extended observations have shown that it occurs most frequently in the choroid plexus and in the ventricles. Progressive growth of the tumor is arrested by fatty degeneration of the tumor cells and by calcification. The tumors, which usually vary in size from a pea to that of a walnut, are often symmetrical, occupying the same location in both sides of the brain. In the lateral ventricles a tumor of fair size may not give rise to any symptoms; in other cases it has caused cerebral disturbances of different kinds and focal symptoms which pointed to the location of the tumor. If the tumor does not undergo calcification, its growth is progressive and it eventually destroys the life of the patient. The clinical history of all such cases is usually one of slow progressive paralysis and death. Among the retrograde metamorphoses in sarcomata, calcification only occurs in psammoma."

Ziegler says "A further peculiarity of sarcomatous as well as fibromatous and myxomatous tumors is the possible formation within the tumor, of circumscribed areas of calcification, which resemble the sand-like particles found in the brain; and from this circumstance some authorities have felt warranted in calling such tumors psammomata (acervulomata, sand-tumors). They are found chiefly though rarely in the membranes of the central nervous system."

Hamilton, a none-the-less eminent authority, says "A psammoma should not be placed among the sarcomata, as undoubtedly it does not belong to them. For want of a better means of classifying

it, the author has included it among the sarcoma-like tumors. It is a tumor of varying size found growing from the choroid plexus, the ventricles of the brain, the arachnoid and dura or pia mater, whose special characteristic is that it contains sand-like concretions. The tumor is made up of a series of endothelium-like cell-nests arranged around blood-vessels, which in course of time become infiltrated with a calcareous salt. It is called an "Angio-lithic sarcoma" by Cornhill and Ranvier on account of its connection with the blood-vessels."

Stengel, an excellent authority, says of the term psammoma, "this represents no distinct species of tumor growth, but rather a peculiarity of different kinds. The name refers to the presence of calcareous matter like that of brain-sand (acervulus cerebri) and psammoma has sometimes been called *acervuloma*. The calcareous matter occurs in the form of rounded masses or concentrically arranged whorls. The tumor-elements themselves may be fibromatous or carcinomatous. In most instances it is angiofibromata or angiosarcomata that present these appearances. Psammomata are met with in the membranes of the brain, the choroid plexus and the pineal gland." In connection with diseases of the reproductive organs he says of ovarian papillary cystomata, "Calcareous bodies arranged in a concentric fashion (psammoma-bodies) are frequently met with in the stroma as well as in the epithelium."

Delafield and Prudden say: "Some forms of psammoma or "brain-sand" found chiefly in the dura mater are fibro-sarcomata which have undergone calcification, the lime being deposited in lamellated masses of various shapes within them."

Without further citations I will proceed with the history of the patient.

CASE 16.—M. S., a woman 74 years of age; mother died, aged 73, of "softening of the brain"—father died, aged 77 years, of "heart disease," one sister committed suicide, another sister died of consumption, and still another sister died of cancer of the breast. As a child the patient was unusually bright and "of a cheerful disposition though regarded by some as rather delicate" —had typhoid fever at 21 years of age but made a good recovery; suffered considerably from "female troubles" all her life, but this complaint never regarded as a serious one.

When 50 years of age is said to have had "a fall down stairs striking on her head" but there were no sequelæ noted, neither was a physician summoned. No history of fracture or period of unconsciousness followed as far as remembered. At the age of 55 she began to develop severe attacks of sick headache and it was first noted that memory was beginning to fail; at the age of 66 she had a severe attack of la grippe and "was mentally unbalanced for some time but finally seemed to recover her previous mental balance;" three years afterward was again taken with la grippe and after recovery from her physical illness did not seem to be balanced mentally and steadily declined after that. Her domestic life was always of the pleasantest until about ten years prior to her mental break-down, when she suffered severe financial reverses and this was given as an assigned cause for her insanity. She never had any children and never had any miscarriages, and there was an emphatic denial of venereal infection.

Admitted to the State Hospital at Waterbury, December 11, 1899 on the following physician's certificate: "For the last two years she has wandered around the streets in an aimless manner, calling at houses without purpose, walking in the middle of the road and asking strangers questions.

"She needs the constant attention of an attendant; she had to be locked in her room nights and rebels against control. She is destructive and has delusions which frighten her and make her more uncontrollable."

On admission to wards was very restless, wandering about in an aimless manner, trying the doors and windows but not giving any reason for so doing; was planlessly resistive and talked a great deal about persons and places she had formerly known but in a garrulous and indifferent manner. Gave evidence of hallucinations of sight and hearing; talked of seeing animals and burglars in her room at night and often showed effect of fear. She said she carried on conversation with persons who had long since been dead and showed considerable pressure of activity during excitement. Her memory was very poor for remote as well as for recent events and she was only partially oriented as to time, place and persons; insight into her mental and physical condition was wholly lacking.

Continued in much the same state as described until the early

part of January, 1900, when she was taken with a slight cough and coryza, which condition in about a week, developed into left lobular pneumonia; expectorated freely but dyspnœa continued quite persistently; was thought to have recovered from this and went about in a rather weakened condition until April 11, when she was again taken with a severe cold, "this developing later into right lobular pneumonia, and died April 14th."

She had been so restless that a more extended examination of her physical status was not made, however there did not seem to be any paralysis of any part of the body at any time.

An autopsy was performed 6 hours and 30 minutes after death and the notes are as follows:

Aged and emaciated, fairly well-nourished, female body; rigor mortis slight, marked post-mortem lividities were present over dependent portion; both feet were in full extension and there was a slight tendency to talipes equino-varus on the left side; no evidence of decubitus was present; arcus senilis distinct; no apparent facial asymmetry but considerable amount of cyanosis was present; the ears being bluish, discolored and the external jugular vein on the right was much distended and unusually distinct (i. e. running from its bifurcation in the supra-clavicular region upward to the inferior maxillary space, this condition was probably a phenomenon due to dyspnœa); the abdomen was much distended and full of gas; the peritoneum was quite firmly adherent to the intestines; the right lobe of the liver lay at least a full inch below the costal margin and was firmly adherent to the surrounding tissues; the diaphragm was also the seat of considerable thickening and had a dime-sized milk-spot upon the middle of the anterior surface; the vessels of the visceral layer of the pericardium were tortuous and generally whitened; the pericardial sac contained about 10 cc. of clear straw-colored fluid.

Heart.—Weight 410 grms. The right side was entirely filled with a large, well-organized clot, with the chordæ tendinæ throughout entwined by it; there were several fairly well-organized clots within the smaller pulmonary vessels; the valves were of normal width but had the ordinary amount of atheromatous deposits thereon.

Left Lung.—Weight 665 grammes.

Right Lung.—Weight 520 grammes. On the left side, the

lower lobe was almost entirely solidified with the exception of a small area near the transverse fissure which appeared œdematous; the apex showed evidence of old scars. The right lung was adherent to the chest wall over its entire surface but particularly firm over the diaphragm, on section showed profuse amount of œdema and congestion but no areas of consolidation.

Liver.—Weight 1500 grammes. Was dissected out with much difficulty, being adherent to diaphragm; the fissure of the gall bladder showed a marked increase in the thickness of the capsule and was traversed by many fibrous strands; a corset sulcus was present in the middle of the right lobe; supernumerary left lobe; the whole organ on sectioning appeared slightly grayish in color and did not show any macroscopic increase of connective tissue.

Spleen.—Weight 100 grammes. Pulpa was unusually soft and the trabeculæ were indistinct. *Pancreas* was normal in appearance.

Left Kidney.—Weight 120 grammes.

Right Kidney.—Weight 110 grammes. The ureter of the right kidney was distended and full of urine, this condition being evidently due to smallness of its caliber near the bladder exit; the pyramids were distinct in both and the capsules were adherent and thickened; the external surfaces of both contained numerous rosettes. *Uterus* and *adnexa* sclerotic. *Ovaries* were adherent to pelvic wall and practically obliterated. *Appendix vermiformis* adherent to surrounding intestines but having no evidence of pus or recent inflammatory products.

Calvarium.—Weight 335 grammes. Symmetrical externally; sutures well preserved; moderate amount of diploe; sulci of blood-vessels rather deep and jagged; about four centimeters to the right of the junction of the sagittal and coronal sutures there appeared a distinctly elevated vertical ridge of bone, the extremities of which were sharply pointed and extended about 3 centimeters in length parallel with the longitudinal sinus into the frontal bone. The dura mater was firmly adherent to the calvarium throughout its entire extent, but was particularly adherent over the frontal area and site of the depression of bone. This upon further investigation was found to be firmly joined to an apple-sized tumor mass extending into the right hemisphere, but apparently not intimately associated with the cortex. See photos

of calvarium and brain showing the association and relation of the above described parts.) In tracing out the dura mater, the tumor was seen to be springing from this membrane and macroscopically the growth appeared of a grayish-red color and was of a firm consistency. The tumor at first sight showed only about a half dollar-sized area, so deeply was it buried within the substance of the cerebrum. Upon further examination the tumor mass was observed to arise distinctly from the dura mater but occupied an area bounded externally by the second frontal convolution, internally by the median raphe with the paracentral convolution or lobule crowded down beneath upon the roof of the corpus callosum; posteriorly by second frontal convolution. The median portion of the tumor presented a deep groove wherein rested the vertical ridge of bone in the calvarium and from this point in the dura radiating fibrous bands were readily seen stretching outward to all parts of the growth. A complete section of the brain at this point showed the tumor mass pressing downward directly upon the internal capsule deflecting the septum pellucidum toward the left and bearing down the corpus callosum on that side; the cinguli gyrus and the superior frontal gyrus being crowded down and apparently atrophied. The caudate nucleus seemed to be the part which had received the most direct pressure, but this, although being somewhat out of place did not appear to be in any way directly altered in structure.

There were no areas of softening present and all of the convolutions seemed well formed. There were no granulations of the 4th ventricle present. Save considerable atheroma of the basilars, the brain otherwise presented few features which would be worthy of notice

Sections were taken of the tumor together with cortex from the paracentral and second frontal convolutions, cerebellum and cornu ammonis from both sides of the brain; the cord, hypophysis and sections of the abdominal and thoracic organs, were also taken and preserved in the usual manner. Nissl and Weigert-Pal staining methods were used for examination of the nervous system and Unna's method was used for staining sections of the tumor and internal organs.

Microscopical Examination: Tumor.—The growth when sectioned seemed very difficult to cut and the edge of the knife

grated some while a section was being taken. The most striking feature concerning the tumor appeared to be its gritty consistency and under the two-thirds and one-sixth objectives there appeared a large number of concentrically arranged whorls or rings of hyaline-like structure, with which various areas of the tissue were literally crowded. These were hardly perceptible to the naked eye, but upon the very closest inspection these spherules appeared elongated but retained the same hyaline, concentrically arranged whorls with a capsule of considerable thickness. Other fields were found where scarcely nothing appeared under the microscope save these particular bodies which it seems are so distinctive of psammomata. Many of these spherules had within their centers a distinctly irregular point of calcareous substance; these of course upon sectioning caused havoc with the usually sharp microtome knife and in most instances caused a tearing of the specimen.

Some of the whorls stained well with Nissl and Unna, while others did not seem to take the stain well at all. As to just what chemical substance these hyaline spherules represent, has not as yet been determined, but it is very evident that the central point is represented by some calcium salt, due to degenerative changes.

A large portion of the growth however consisted of small spindle-cells with elongated oval nuclei with very little intercellular substance separating them, and in other areas there appeared a decidedly fibrillated appearance and in still other fields there were beautifully curved bands running about the hyaline spherules.

There was hardly any evidence of blood-vessels in the growth and very little connective tissue present, as there appeared to be a gradual transition from connective tissue in the dura mater to the spindle-celled structure of the tumor together with its decidedly psammomatous characteristics.

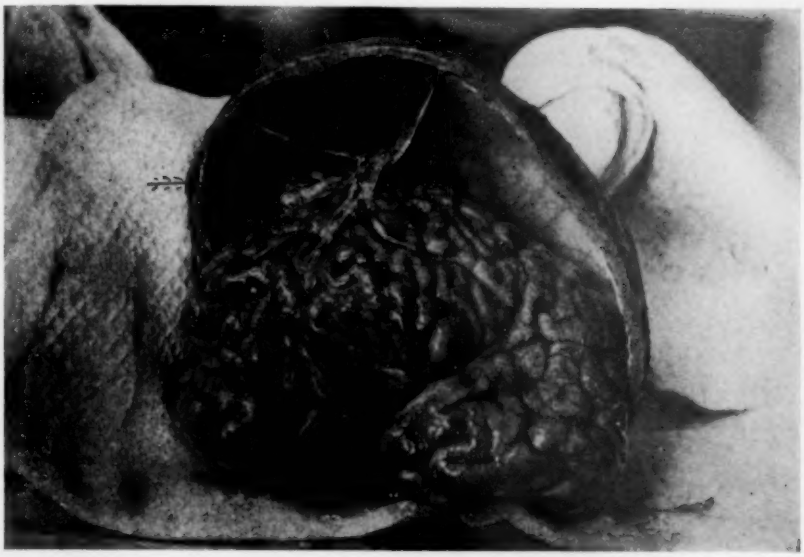
In general the cells of the liver were somewhat atrophied but the viscus was not specifically cirrhotic. The sections made from the other organs showed few cell changes worthy of note.

Weigert-Pal sections from the medulla and upper portion of the cervical cord showed no tract degenerations.

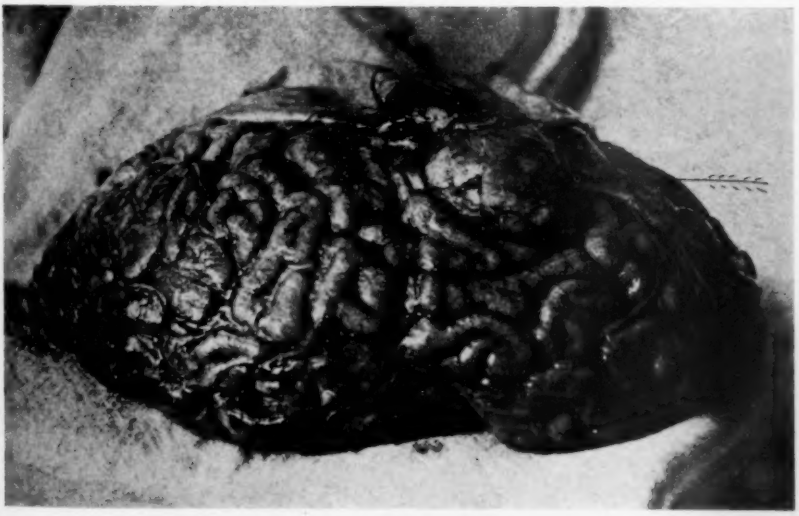
In conclusion then, the interesting and peculiar feature of this case are: *first*, that no apparent paralysis or interference with co-ordination and locomotion occurred, although there was present a large growth directly imbedded in the convolutions and thereby

causing considerable pressure; *second,* the malignant growth was presumably of traumatic origin, springing from the dura mater but principally caused by the vertical ridge of bone pressing downward upon the meninges; *third,* that no evidence existed externally of a depressed fracture of the skull.

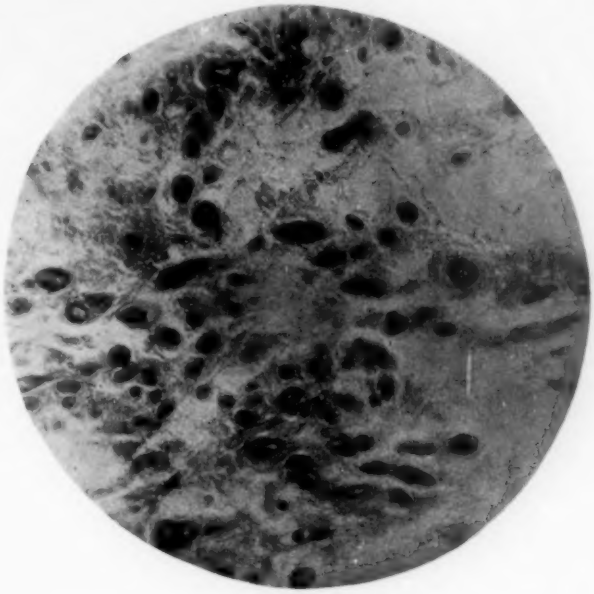
In connection with the preparation of the necessary tissues for microscopic study, I wish to acknowledge the help of my assistant Mr. Herman D. Bone, and in reference to the photo-micrographs, I wish to acknowledge the work of Dr. M. J. Wiltze, of the State Laboratory of Hygiene, who has made it possible for me to illustrate this contribution to the literature on the subject.



Psammo-sarcoma as it appeared attached to skull.

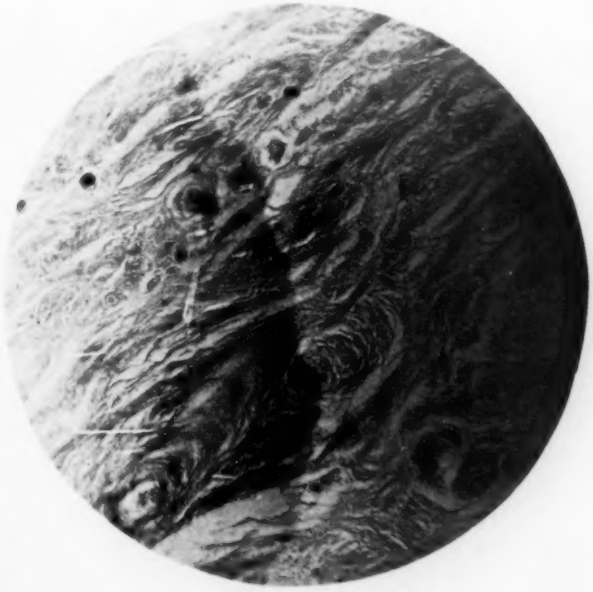


Psammo-sarcoma as it appeared after removal from the cranium.



× 50

Showing large number of psammoma bodies or whorls of various shapes and sizes. Nissl stain.

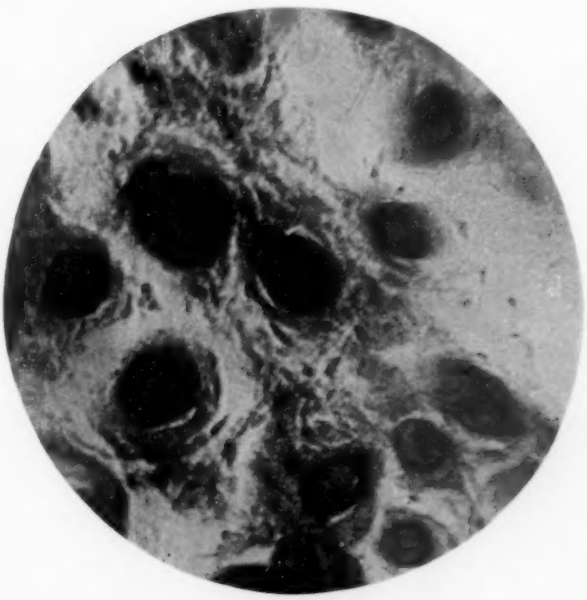


× 50

Stroma and psammoma bodies. Unna stain.

× 330

Showing spindle cell structure and a few spherules—fibrous stroma. Unna stain.



× 330

Showing psammoma bodies or whorls and their concentric arrangement. Nissl stain.

Notes and Comment

JOURNAL DE PSYCHOLOGIE.—It is with no little pleasure that we welcome the *Journal de Psychologie normal et pathologique,* the initial number of which is for January-February, 1904. The reputation of the editors, Dr. Pierre Janet and Dr. Georges Dumas, is sufficient guarantee of its excellence, but the table of contents and list of promised papers leave no doubt that the journal will be conducted upon a high plane. The first number contains four original articles (pp. 1-37), and 84 abstracts from psychologic literature (pp. 38-112) which are so divided into subgroups that they are convenient for reference. We extend to this journal our sincere congratulations and wish for it a long and prosperous career.

JOURNAL OF PHILOSOPHY, PSYCHOLOGY AND SCIENTIFIC METHODS.—Somewhat more general in scope than the above journal is the *Journal of Philosophy, Psychology and Scientific Methods,* a fortnightly, the first number of which was published January 7, 1904. It is edited by Professor Frederick J. E. Woodbridge, of Columbia University. In the first article by Professor Münsterberg, on the International Congress of Arts and Science, the scope of the journal is defined as follows:—"It desires to stand for the unity of knowledge, aims to consider the fundamental conceptions which bind together all the specialistic results, seeks to enquire into the methods of science which bind together the scientific workers, and into the center of its sphere it puts philosophy." With such aims it would appear that the journal will take a high plane. Besides the above article there is one by Professor Ladd, a discussion by Christine Ladd Franklin, a society report, several reviews, and notes of interest, all occupying 28 pages. To this journal we also extend cordial wishes for a successful career.

AMERICAN MEDICO-PSYCHOLOGICAL ASSOCIATION.—As heretofore announced the American Medico-Psychological Association will hold its next annual meeting in St. Louis, May 30 to

June 3, inclusive, under the presidency of Dr. A. E. Macdonald, of New York.

The local committee has wisely decided to hold morning sessions only. Under these circumstances the allurements of the Exposition ought not to detract from the scientific value of the meeting or distract the attention of members. Special railroad rates will doubtless be obtainable, and hotel accommodations at reasonable prices (Planters Hotel) have been provided for.

The addresses of welcome will be given by His Excellency, A. M. Dockery, Governor of Missouri, by the Honorable Rolla Wells, Mayor of the City of St. Louis, and on the part of the medical profession of Missouri by Dr. Wm. G. Moore, President of the State Medical Association.

The annual address will be delivered by Dr. C. G. Chaddock, of St. Louis.

Papers have been promised as follows:

"Amnesia Clinically and Diagnostically Considered," Chas. H. Hughes, M. D., St. Louis, Mo.

"A Plea for the Voluntary Admission of Certain Types of Insanity in Institutions for the Insane," James Russell, M. D., Hamilton, Ont.

"A Consideration of the General Conditions Associated with Insanity and their Connotation Statistically and Otherwise," H. A. Tomlinson, M. D., St. Peter, Minn.

"Tubercular Meningitis in the Adult," Frank P. Norbury, M. D., Jacksonville, Ill.

"A Case of Sleep Talking," D. R. Burrell, M. D., Canandaigua, N. Y.

"The German Psychiatric Clinics," E. N. Brush, M. D., Towson, Md.

"Surgical Work Among the Insane," A. T. Hobbs, M. D., Guelph, Ont.

"The Alcoholic Psychoses," Henry P. Frost, M. D., Buffalo, N. Y.

"The Mental Conditions Occurring in Cretinism," Edward E. Mayer, M. D., Pittsburg, Pa.

"Are the Insane Responsible for Criminal Acts?" John Punton, M. D., Kansas City, Mo.

"The Epileptic Child: Its Treatment and Care," W. P. Spratling, M. D., Sonyea, N. Y.

"Organic Dementia; with Abstract of Fifty-Eight Cases," J. M. Keniston, M. D., Middletown, Conn.

"A Border Line Case," C. Eugene Riggs, M. D., St. Paul, Minn.

"The Variations of the Psychic Equivalent," F. Savary Pearce, M. D., Philadelphia, Pa.

"Some of the Medico-Legal Relations of Paranoia," Chas. K. Mills, M. D., Philadelphia, Pa.

"A Few Remarks about Observation Hospitals and Wards," E. Stanley Abbot, M. D., Waverley, Mass.

"Suicide and Insanity," G. H. Hill, M. D., Des Moines, Ia.

"Reconciliation of the Disparity between Hospital and Asylum Trained Nurses," C. P. Bancroft, M. D., Concord, N. H.

"Suicide and Insanity," Chas. W. Pilgrim, M. D., Poughkeepsie, N. Y.

"The Therapeutic Influence of Occupation and Diversion in Insanity," Chas. E. Atwood, B. S., M. D., White Plains, N. Y.

"A Review of the Recoveries of the St. Lawrence State Hospital in the Year 1894," Richard H. Hutchings, M. D., Ogdensburg, N. Y.

"The Mental Results of Abdomino-Pelvic Operations in Insane Women," W. P. Manton, M. D., Detroit, Mich.

"Extension of Tent Treatment to Additional Classes of the Insane," C. Floyd Haviland, M. D., and Chester Lee Carlisle, M. D., Ward's Island, N. Y.

"Intra-Cranial Tumors in the Insane; with a Report of Two Cases," I. H. Neff, M. D., Pontiac, Mich.

"A Remarkable Case of Degenerative Insanity of the Moral Type," Henry R. Stedman, M. D., Brookline, Mass.

"A Medico-Legal Case of Well-Poisoning—With a Plea for a Hospital Observation Law," Henry R. Stedman, M. D., Brookline, Mass.

Papers of which the titles cannot now be announced have been promised, or provisionally promised, by Byron M. Caples, M. D., Waukesha, Wis.; James H. McBride, M. D., Pasadena, Cal.; Chas. G. Wagner, M. D., Binghamton, N. Y.; C. R. Woodson, M. D., St. Joseph, Mo.; Dwight S. Moore, M. D., Jamestown, N. Da.; Wm. A. White, M. D., Washington, D. C.; Richard Dewey, M. D., Wauwatosa, Wis.; Geo. C. Crandall, M. D., St. Louis, Mo., and Carlos F. MacDonald, M. D., New York, N. Y.

Obituary

EDWARD CHARLES RUNGE, M. D.

Edward C. Runge died February 10, 1904, in his forty-eighth year, at St. Louis, Missouri, after an illness so brief that the announcement of his death came like a shock to his friends and the community at large.

Dr. Runge resigned the superintendency of the St. Louis City Insane Asylum February 1, 1904. For many months previous to his resignation, to his duty as head of a much over-crowded institution, always wearing both physically and mentally, were added the annoyance and strain of attempting to carry on his work in the face of persistent official interference with his established methods of administration. If there had been a desire to force Dr. Runge to resign, more effectual means could not have been devised. Had his resignation been demanded, we do not question that it would have been immediately tendered. It is sad to think that an early request for his resignation might have spared the life of a zealous public servant, a true citizen, a distinguished physician, whose life-work was that of devotion of his talents and strength to a most unfortunate class, without thought of personal gain.

Worried and worn by months of conflict, at a time when he should have been in a sick-bed, Dr. Runge spent several days of most painstaking effort to acquaint his successor with the management of the asylum, and especially with the patients in his charge; his highest duty seemed to him to be to have no disadvantage come to his patients from his act of leaving them. Thus suffering in mind and body, he fell an easy victim to pneumonia; he seemed to have no chance of recovery from the moment he was stricken.

It seems reasonable to ask why Dr. Runge did not resign much earlier, and thus save himself the strain which contributed to sac-

rifice his life. Strong in his conviction that the indigent insane of a community should not be subject to the needs of politicians; that those who cared for them should be chosen by reason of fitness rather than for political views or party services, he felt that he should do all in his power to protect those for whom he had labored so long and successfully from the dangers he believed to be threatening them. At last convinced that his ideals could not serve as an inspiration to those in authority, and seeing an unending conflict ahead, he felt he could do more for his charges by leaving them.

The esteem in which Dr. Runge was held by all of those who knew him or came in contact with him, has found expression in the organization of a representative committee with the object of holding a public memorial meeting to commemorate his public services, and to provide some permanent memorial of him and his life-work.

Dr. Runge was born in St. Petersburg in 1856, of German parents. He received a careful preliminary education, but his study of medicine was prevented by the death of his father and the consequent need to provide for his mother and numerous younger children of the family. After he had aided his family to independence, at the age of twenty-six, with no knowledge of English, he came to America. Devoid of means, but confident, he accepted the first place offered him, that of baggageman at the Union Station in St. Louis. Here his acquaintance with modern languages (he spoke Russian, German, French, and had a useful knowledge of Italian and Spanish) helped him to public notice, and with the betterment of his material position, he was able to realize his early ambition to study medicine. He graduated from the St. Louis Medical College (Washington University) in 1891. His student years were hard and would have been discouraging to one not possessed of untiring energy and will. He fortunately obtained the aid of a scholarship, and his studies were completed by working as a night-clerk and as a Pullman car conductor. During his student days he was closely associated with Professor Porter, of Harvard, at that time professor of physiology in the St. Louis Medical College, and did special work in physiological chemistry. His first year after graduation was spent as an interne at the Female Hospital; he then became as-

sistant to Dr. Frank R. Fry, professor of nervous and mental diseases in the St. Louis Medical College, and continued to fulfill this function with his private practice until his appointment as superintendent of the City Insane Asylum in May, 1895. He was later appointed Lecturer on Psychiatry in Washington University, but relinquished this place a year before his death.

Dr. Runge became a member of the American Medico-Psychological Association in 1896, and was thereafter an active and interested worker at its meetings. It is not too much to say that his positive, enthusiastic personality, with its marked idealism, was one to be remembered and to win the respect and affection of his associates. His papers before the Association were always interesting and suggestive, breathing something of the freshness and freedom from prejudice so characteristic of the man. He did not attend the last meeting of the Association; his place as superintendent, until then one of mere medical endeavor and administration, had become a source of worry.

The following quotation expresses most fittingly the debt of St. Louis: "The service that Dr. Runge has done for St. Louis, especially, and for other municipalities with the same sort of problem in the care of the insane, may be known by considering the institution, the Insane Asylum, before and after he became superintendent. It was built originally to accommodate three hundred patients; there were no adequate means of making room for more; there was no equipment worthy of the name, and the traditions left behind were of the most antiquated and narrow. In eight years there were in the asylum at all times over seven hundred patients, and within the narrow limits which were imposed by the municipal powers the place had changed and had become the modern insane asylum. All this had been done, and no one but those who know can appreciate what this means, without any additional equipment that amounted to anything and without an increase in the medical or administrative body. This means that Dr. Runge's personality had so filled the institution he controlled that it reflected his own spirit in every one of the departments, and nowhere was this better shown than in the attitude of the patients themselves toward the superintendent and the institution."[1]

[1] Sidney I. Schwab, M. D., Interstate Medical Journal, March, 1904.

During the eight years of Dr. Runge's incumbency, the writer had the honor and profit of frequent contact with him in his work, circumstances which gave the privilege of appreciating the rare qualities of the man and his friendship. His unfailing kindness and the active interest he took in placing all possible clinical material at the disposal of teachers for practical lectures on psychiatry, are to be mentioned. He did all he could to further the teaching of the science to which he was wedded. On more than one occasion, and, indeed, for an entire session, he consented to give clinical instruction in psychiatry to students of a medical school with which he was in nowise connected.

The writer's acquaintance with Dr. Runge began in 1895 in connection with clinical lectures at the asylum, and then and thereafter was noted what seemed very remarkable as indicating his unusual ability: without previous "asylum" training and with a comparatively limited practical acquaintance with insanity and insane patients, he quickly showed his thorough comprehension of the problem he had to solve; he was a master from the first—a born alienist and administrator of a hospital. It would seem that his freedom from asylum traditions and prejudices made it possible for him to accomplish with astonishing rapidity the modernization of an antiquated institution, while hampered extremely by lack of funds and adequate room.

To the devoted wife, Emily Foote Runge, who through ten years lightened his labors with her unfailing sympathy and intelligent material aid, may some comfort come in the thought that the good he did remains; that she is not alone in sorrow and regret!

CHARLES GILBERT CHADDOCK.

Book Reviews

The American Year-Book of Medicine and Surgery for 1904. Vol. I. General Medicine. Edited by GEORGE M. GOULD, A. M., M. D. (Philadelphia, New York, London: W. B. Saunders and Company, 1904.)

This work continues to show the same degree of excellence as heretofore. The summaries of the recent literature of the different specialities and diseases are made briefly, but clearness has not been sacrificed. This sort of book is of value, both to the busy practitioner who has but little time to keep pace with the literature of general medicine and the specialties, and to the specialist who desires to broaden his knowledge to limits outside of his own particular field of study. The section devoted to Nervous and Mental Diseases is under the immediate charge of Dr. Archibald Church who states that "in the matter of mental diseases very little of importance has appeared during the year." This section comprises 38 pages of which 4 are devoted to mental diseases, and contain abstracts from eight articles only. It would seem that much more important papers have appeared than some of those abstracted and that the number has been so curtailed that these 4 pages are of little value to one who desires to inform himself as to the advances which have been made in psychiatry. The objection that the articles abstracted form but a small part of all which have appeared on the subject applies throughout, and the book would gain in value were references given to papers which have not been abstracted. W. R. D.

Die Progressive Myoklonus-Epilepsie (Unverricht's Myoklonie). Von Dr. HERMAN LUNDBORG, privatdozent der psychiatrie und neurologie in Upsala. (Herausgegeben mit unterstutzung aus Wilhelm Ekman's Universitatsfonds.) Progressive Myoclonus-Epilepsy.

This is the third monograph the author has given us upon this interesting association disease. It is the most comprehensive and complete exposition of the disease in any language. The nature, pathology, cause, symptoms, prognosis and differential diagnosis of the affection are given most minute attention. Every phase and stage of the affection are detailed at length from all view-points. The different forms of the disease and atypical cases are illustrated. As the nature of this disease entity is now quite established the etio-pathology and pathology press for solution. The author is inclined to believe as Clark and Prout do, that its cause is one of auto-intoxication. Lundborg especially emphasizes the possible connection of the disease with perverted metabolism of the ductless glands, as Ohlmacher holds for epilepsy proper.

The varying phases of etiology, symptomatology, diagnosis, prognosis and treatment are excellently portrayed from the many recorded cases. The differential diagnosis is especially interesting in that the close relationship of a number of convulsive disorders is considered, such as Huntington's chorea, Koschewniskow's epilepsy, hystero-epilepsy, electric chorea (Henoch-Bergeron), paralysis-agitans and myotonia, fibrillary chorea (Morvan) and myokymie (Schultze). While Lundborg believes all have a kindred pathology in the cortex, he states that there may be, and indeed are, evidences of secondary attendant lesions in the cord and periphery.

The 229 works of reference would serve excellently as a bibliography of the entire subject. On the whole we may say that this excellent monograph is the best and most complete in any language upon the combination disease.

L. Pierce Clark.

Pamphlets Received

Reports of the Trustees and Superintendent of the Butler Hospital, presented to the corporation at the sixtieth annual meeting, January 27, 1904, Providence, R. I.

Flatulence, Meteorism and Tympanites. Charles D. Aaron, M. D. Reprinted from the Dietetic and Hygienic Gazette, August, 1903.

Treatment in Four Hundred and Forty-Two Cases of Movable Kidney Without Surgical Intervention. Charles D. Aaron, M. D. Reprinted from American Medicine, Vol. VI, No. 23, pages 893-895, December 5, 1903.

Third Annual Report of the New York State Hospital for the Care of Crippled and Deformed Children for the year ending September 30, 1903.

Annual Report of the Surgeon-General of the Public Health and Marine-Hospital Service of the United States for the fiscal year 1902.

Sterile Water Anæsthesia in the Office Treatment of Rectal Diseases. Samuel G. Gant, M. D. Reprinted from the New York Medical Journal and Philadelphia Medical Journal, January 23, 1904.

The Principles of Diagnosis of Medical Malingering. John Punton, M. D. Reprint from the Medical Record, January 9, 1904.

Annual Report of the Managers of the Western Pennsylvania Hospital, the Department for the Insane at Dixmont, 1903.

State Care of the Insane. By Dr. George J. Preston.

Eighth Annual Report of the Board of Managers of the Springfield State Hospital of the State of Maryland, Sykesville, Maryland, to His Excellency the Governor of Maryland. October 1, 1903.

Successful Measures for the Prevention of the Spread of Smallpox, Adopted at the New York State Reformatory at Elmira, N. Y. Frank W. Robertson, M. D.

What is Epilepsy? J. W. Wherry, M. D. Reprinted from American Medicine, August 15, 1903.

The Significance of the Convulsion in Idiopathic Epilepsy. J. W. Wherry, M. D. Reprinted from American Medicine, September 5, 1903.

Results of an Experimental Treatment of Epileptic Insanity. J. W. Wherry, M. D. Reprinted from American Medicine, January 9, 1904.

Fifteenth Annual Report of the New Jersey Training School for Feeble-minded Girls and Boys.

Thirteenth Biennial Report of the President and Visitors of the Maryland School for the Deaf and Dumb to the Legislature of Maryland, from October 1, 1901, to September 30, 1903.

New York State Reformatory at Elmira. Report of the Board of Managers, Report of the General Superintendent, and the Financial Statement for the fiscal year ending September 30, 1903.

Are we to have a United States Medical Profession? Charles S. Mack, M. D.

The Development of Obstetric Surgery. James U. Barnhill, M. D. Reprinted from Columbus Medical Journal, August-September, 1903.

Half-Yearly Summary.

MASSACHUSETTS.—*Worcester Insane Hospital, Worcester.*—The nurses' house, begun in May, 1902, is now practically completed and will soon be occupied. It is located on the right and in front of the main building, just off the main driveway. It is built of stone from the hospital quarry, with brick trimmings, to conform architecturally with the other hospital buildings. It is three stories in height, with a high basement. It consists of a central building, 40 feet long by 46 feet wide; and two wings, 48 feet long by 37 feet wide, making the entire front 136 feet. On the first floor of the center building, on either side of the main entrance, are the reception room and the office of the superintendent of nurses. Directly over these, on the second floor, is a class room 14 feet wide by 25 feet long; while in the rear, on each story, across a wide central corridor, are the toilet rooms. Here also is located the main staircase, which is enclosed in brick walls and shut off on each story by swing doors. Adjoining the staircase and in the same enclosure is an elevator shaft, also of brick. A room is provided in the second story for the cooking school, and one in the third story for the housekeeper. In the two wings, besides a linen closet on each floor and a staircase at either end, there are 53 single rooms, 9 feet 6 inches wide by 14 feet long, and three double rooms, for nurses. The building is to be lighted by electricity and heated by steam from our central heating plant, by the direct, single-pipe system.

Although exceedingly plain, both in exterior and interior finish, it is well adapted for the purpose for which it was designed, and in every way serviceable. All of the rooms are bright and sunny, and all, both on front and rear, have a delightful outlook over the hospital grounds and the surrounding country.

The hospital is so open to inspection that all who are interested, whether the general public or the relatives of the patients, can be well informed as to the condition of the inmates, the care and attention they receive and the methods by which the institution is managed. Wednesdays and Fridays, from 10 A. M. to 4 P. M., are visiting days, when the wards are filled with friends of the patients, who have an opportunity to see them in their every-day life and routine. Meetings of the Board of Trustees are held each month, when accounts are audited and financial conditions examined, wards are visited, patients seen and listened to, bedding and clothing are inspected, food sampled and the mode of its preparation noted. Besides this, individual members of the Board make unexpected visits at odd times, both by day and night, when the regular work of the house can be care-

fully studied. Visits are also made in company with the officers of the hospital on Sunday mornings, when both patients and attendants are expected to present themselves in their best attire. At that time the rooms, the closets, the bedding, and in fact every corner of the house is thrown open for inspection. *An intelligent interest by the outside public in the delicate and difficult work that is being done in this large and pitiful community would be a positive help and inspiration to those who are devoting themselves to its accomplishment.*

—*Taunton Insane Hospital, Taunton.*—The pathologist's report for the year shows activity in medical work. In the clinical department a more scientific and accurate study and registration of the all-important clinical manifestations is constantly sought. Efforts in this direction are bearing fruit in the stimulated interest in the work and the increasing value of the work done.

The organization of the staff to this end is as follows: Each service, male and female, has two senior physicians. The cases on admission are assigned in rotation, and the physician to whom the patient is assigned then becomes responsible for the mental and physical examinations in the manner indicated in the last report. Two internes are engaged in the clinical work, one in either service, who work under the guidance of the senior physician and pathologist. The pathologist has general supervision over the clinical records. A third interne is retained as a laboratory assistant, and the other internes are given opportunity for work in that department. Much assistance is received from the nurses, who are given instruction on the observation of patients, and are offered every opportunity to apply such instruction.

One of the staff conferences each week is taken up with the report of literature, a generous supply of which is allowed by the Board of Trustees. The various periodicals are divided among the members of the staff, who take their turn in presenting an abstract of the interesting and valuable contributions.

—*Danvers Insane Hospital, Hathorne.*—This hospital has practically completed a group of colony buildings at a distance of one mile from the main hospital. These buildings have been designed with the view of accommodating quiet chronic female patients. The general plan is that of one story wood buildings, with large day rooms and large dormitories, comparatively few single rooms being provided. No guards have been placed upon the windows as only quiet patients will be kept there. A single large dining-room, in the center of the group, from which covered corridors radiate to the several buildings, will accommodate both inmates and employees. An independent steam plant has been provided with a capacity to heat one or more additional groups of the same size as those built this year, as further additions become necessary. Buildings will be lighted by electricity generated at the main hospital. A separate medical officer will not be stationed at the new buildings at present. The present

number of inmates is 1256 and it is expected that one hundred additional inmates will be sent as soon as they can be located at the new buildings, probably in a month.

—*State Farm at Bridgewater.*—During the year certain extensions and improvements of the institution plant, as authorized by act of legislature, have been in progress. There were appropriated $8600 for a new bakery and for the roofing of the hospital, and $7400 for a boiler and for laundry machinery. The improvements contemplated in the appropriations are in progress and well on towards completion. Under chapter 414, Acts of 1903, $100,000 was appropriated for the erection of a new ward in the Asylum for Insane Criminals. The building thus authorized is in process of erection. The farm area has been somewhat extended during the year, in the use of money appropriated for that purpose, as stated by the superintendent in his report to the trustees.

The work of subduing rough lands and bringing them into cultivable conditions, which has long been a valuable feature of the life of this institution, has gone forward with the usual good results.

—*State Hospital at Tewksbury.—Improvements made during the Year.*—A new pump of 1,500,000 gallons capacity per twenty-four hours has been placed in the pumping station, and the old one repaired so that there will be no future danger of a shut-down in that department. A new connection with additional shut-offs has been made just below the standpipe, so that in case of diminished pressure water could be forced directly from the pumping station into the supply pipes around the institution. A new underwriters fire pump with capacity of 750 gallons per minute has been set up in the engine room, with supply pipe from a group of old cisterns in the center of the yard, and with delivery pipes running to hydrants, giving much additional protection in case of fire. Continuing the work on the cold storage plant, a compressor and pump have been installed and have been in operation since last spring. After a certain amount of experimentation and operation for the sake of clearing the pipes, satisfactory results have been obtained. Regular refrigeration in the large storage rooms has just begun. The mortuary chapel has been completed and furnished, and is greatly appreciated by friends of the hospital and of the deceased patients. An extension to the women's dormitory has been erected, and is now near completion. It is two stories in height, and has a good, light basement. The first floor will furnish additional dormitory space and a sitting room for the aged women. On the second floor will be a children's dormitory and also day room. In the tower at the extreme end is an iron fireproof staircase, and the sanitary sections for each floor.

A surgical building has been erected in the south-east corner of the main court, between the women's asylum and the men's hospital; the partitions have been erected and the building is now ready for plastering. This building will contain an operating room, two recovery or etherizing rooms, a wide corridor, sterilizing rooms, lavatory and surgeon's room. It is built

of brick, with terra-cotta partitions, iron girders with cement arches for supporting the floor. Furniture for the asylum for insane men has been made during the year.

—*Massachusetts Hospital for Dipsomaniacs and Inebriates, Foxborough.* —At the close of the first decade of the operation of the institution, an inquiry has been made into the results of treatment. Early in the year the work was begun of gathering data concerning all regularly discharged patients. A circular letter was sent to all cities and towns except Boston, where special agents were employed, with a list of patients, giving the date of admission and the date of the last discharge from the opening of the hospital up to January 1, 1903, thus securing a period of uninterrupted absence from the hospital of from six months to nine years in some cases. The chiefs of police and other officials have given generous aid in this research. The results are very gratifying, and it would seem as if there could be no further question of the usefulness of the hospital.

It has been impossible, in the time given to the work, to trace out the histories of all these men; this applies especially, as might be expected, to the earlier years; but enough has been learned to clearly demonstrate the need of the hospital, and that its work is of value.

The number of persons discharged as previously described is 1043; of this number, 243 are known to be temperate or abstinent,—23 per cent of the whole; 206 are improved, or 19 per cent of the whole. Thus, of all those who received the full course of treatment, 42 per cent were either cured or improved, while the other 58 per cent include the 108 who died and the 336 who could not be found, some of whom it may be fairly assumed have been benefited. Many more might be properly included among the "improved," as there have been many instances where men have been abstinent for one, two, three, even six or more years, before again seeking the aid of the hospital; but they could not be credited with the full amount of benefit really due them from the hospital under the rule which takes from last discharge only.

The patients who did not receive a full course of treatment must include all those who were discharged by the trustees on account of unfitness, and all those who, after staying for a time, discharged themselves by elopement and were not returned; as has been explained, these classes were much larger in the early years of the hospital, before the magistrates had learned by experience to select prompt cases, and in respect to which there has been an increasing improvement in the last few years.

—*Massachusetts Hospital for Epileptics, Palmer.*—Several new buildings have been completed during the past year, and are now wholly or partially in use. The nurses' home, which a year ago was in process of construction, has now been finished and furnished, and the nurses have taken up their rooms there. This building has 40 single rooms.

The new laundry building is a large, one-story brick structure, 120 feet long by 60 feet wide, giving ample facilities for the present work and for growth. This also has been completed during the past year.

The third building built and completed during the past year is the electric power house, which is directly connected with the old boiler house, and affords the necessary room for dynamo and engines. It seems to be well constructed and adapted to needs, and should afford the space required for all the machinery requisite for power and the lighting of the present groups of buildings, and for future extensions.

In addition to the completion and furnishing of these buildings, an adjoining farm of 300 acres, with a good brick house and several barns, has been purchased. On this land on the hillside opposite the existing buildings two suitable buildings are to be erected, to contain 60 patients, and these are, with the brick building and barns already existing, to form a farm group. These new buildings are now in process of erection, the foundations have been made, and the framework is nearly complete.

The number of patients in the hospital October 1, 1902, was 377; on October 1, 1903, it was 422,—an increase of about 12 per cent. As a result of this, the institution is beginning to be overcrowded; 50 patients are now obliged to sleep on movable beds made up in corridors and day rooms.

MICHIGAN.—*Eastern Michigan Asylum, Pontiac.*—During the past year the following improvements have been in progress:

The water-supply of the asylum has been increased by the construction of an additional deep-well, similar to the one previously in use.

The central heating-plant has been changed by the substitution of new vertical boilers for the old horizontal boilers which have been in commission since the opening of the institution, by the enlargement of the boiler-house and coal-shed, by the installation of a water-purifying system, and by a complete rearrangement of the heating equipment in the boiler-room.

Various changes have been made in the shops of the institution to meet the growing needs of the asylum for more room and greater convenience.

MISSISSIPPI.—*East Mississippi Insane Hospital, Meridian.*—The last legislature authorized the erection of two new buildings, but failed to make an appropriation for furnishing them, or to provide for the increase in the number of patients. For this reason the erection of the buildings was deferred until this last summer. The buildings are well under way and will be finished in November.

The building for women is a one-story cottage, with capacity for fifty. It has one large and one small associated dormitory, and several small single rooms. The day-room and the dining-room are both large, airy and well ventilated. The toilet and bath-rooms will have both bath tubs and shower baths. This building will be used for the sick and infirm, and the large galleries, in front and rear, will give those who are not able to go out an opportunity of getting fresh air and sunshine. The front porch is 30 x 30 feet, and the rear gallery is 14 x 20 feet.

The building for men is two stories, and in the shape of a cross, with a rotunda and cupola in the center for ventilation. Two wings are divided into wards containing twelve rooms each, and each ward can be isolated.

The other wings on the first floor will contain the dining- and day-rooms for all the patients in this building, on the second floor, this space will be used for associated dormitories. There will be toilet and bath-rooms on both floors, and the bath-rooms will be equipped with both tubs and shower baths.

These buildings are neat in appearance, and well built. The outside walls are of hard brick, uniform in appearance; the inside walls will be finished with acme cement, and the wood work of hard wood, oil finished. The roof is of slate.

NEW HAMPSHIRE.—*New Hampshire State Hospital, Concord.*—The legislature, at its biennial session in 1903, passed an act providing for State care of all the dependent insane. By the provisions of the act all the dependent insane at present requiring remedial treatment are to be committed to the State Hospital at Concord, there to be supported at the expense of the State. Beginning with the year 1905 transfers to the State Hospital of the dependent insane at that time confined in the various county almshouses are to be made pro rata to the population of the various counties as rapidly as suitable provisions can be made for them at the State Hospital. This gradual withdrawal is to continue until the year 1909, when it is expected that all the dependent insane will be transferred to State care. In the meantime no further provisions for the insane are to be made at the almshouses.

The same legislature appropriated $60,000 for additional wings for the quiet demented insane. These new additions are nearly completed. They are two-story structures, slow burning, so-called mill construction. The first floor is a day-room; the second floor a dormitory. The finish is ash. Each day-room has a large sun-room facing the south and west. In these wings will be cared for all such demented insane as require personal night supervision. The passage of the above act marks the conclusion of a long struggle in New Hampshire for the better care of the dependent insane. For years the elder Dr. Bancroft labored for the attainment of a more humane and systematic care of the indigent insane. The good work instigated by him has at last, after nearly thirty years, reached consummation in the legislative State Care Act of 1903.

The affiliation of the Training School for Nurses with the Concord District Nursing Association, commenced in January, 1902, has proved of great value to the nurses. The latter are brought into closer touch with general nursing and such affiliation rounds out their hospital experience in a most practical way. By reason of this work in district nursing in the city of Concord a great step is made toward reconciling the disparity between general and special hospital training.

Dr. H. C. Goodwin, formerly assistant physician at the State Hospital at Tewksbury, Mass., has served as first assistant physician during the prolonged absence of Dr. F. L. Hills, who returns April 1. Dr. G. H. Maxfield, also from the State Hospital at Tewksbury, has taken the second assistant physician's position as well as that of pathologist. Dr. C. H. Dolloff has served as third assistant physician in the wards and laboratory.

NEW YORK.—*The Society of the New York Hospital, Bloomingdale, White Plains.*—There were 29 more admissions to Bloomingdale this year than last. The average number of patients was 336,—7 more than the average of the preceding year.

The number of weeks' board and treatment furnished for patients was 17,525, an increase since last year of 397 3-7 weeks. The average expenditure per patient was $12.21 per week, which includes the total cost of maintaining the property at White Plains and the patients upon it, both insane and at the convalescent homes, for all salaries and wages, repairs, supplies and maintenance of every kind. This was a decrease of 7 cents per capita of the cost of the preceding year.

The total expenditures for the year were not materially increased over those of the preceding year, notwithstanding that articles of current consumption of almost every kind cost more, and the unusually cool and wet summer caused a material reduction in the products of the farm and garden, and although the institution supported an average of 10 more insane patients, and 33 more convalescent patients from the New York hospitals.

Sixty women were beneficiaries of the John C. Green Memorial Fund during the year 1903. The amount of income received from that fund and expended for these women in 1903 was $6842.51, a decrease from the income of that fund below the year 1902 of $113.63. The total charitable expenditures during the year 1903, in caring for and treating patients (preferably acute and presumably hopeful cases), was $54,830.02. Two hundred and fifty-nine individuals shared in this charitable assistance, some being entirely and others partially supported. The sum of $47,987.51 was expended for this purpose from the earnings of Bloomingdale during the year 1903, from which sum the charitable assistance to patients, both insane and convalescents, from the New York hospitals, was given to the extent mentioned above, all profits from remunerative patients being expended in maintaining the institution, and supporting patients not able to pay remunerative prices. 8 per cent of the patients treated contributed nothing whatever to their support; 57 per cent of the patients paid sums varying from nothing to the bare cost of their care. Only 43 per cent paid a rate which secured a profit to the institution. The total support and the partial assistance rendered to patients unable to pay for their entire cost to the hospital was equivalent to maintaining 86 free beds.

There have been no changes in the staff during the year, excepting that made by the ending of the term of Dr. F. M. Cunningham on April 1, 1903, as clinical assistant physician, who was succeeded on April 1, 1903, by Dr. Lemuel S. Allen, Dr. Allen being succeeded by Dr. L. M. Rea on November 1, 1903, who remained until December 10, 1903.

Training School.—The institution was visited on the 16th day of May by the president, vice-president, treasurer, governors of the society and members of the Bloomingdale committee, who made an inspection of the buildings and grounds and found the appearance and condition good, on which occasion they attended the exercises in which the graduates of the Bloomingdale Training School for attendants were given their certificates

and medals. The graduates, including the six who graduated this year, amount to a total of 68 since the school was first started in the year 1895, of whom 12 men and 25 women remain in the employ of the institution.

Outings.—Besides the regular outings, the summer excursions were maintained to the Oakland Beach at Rye throughout the summer, where the hospital has hired a small pavilion, whenever the weather was suitable; also a number of excursions were made by men patients and their attendants on the trolley cars to Mamaroneck, and thence on the sound in a naphtha launch. As these excursions were a variation of the ordinary routine, they seemed to be greatly enjoyed by those who participated in them.

All the regular amusements and outdoor exercises like tennis, base ball, golf, etc., were maintained with interest throughout the season.

Convalescent Homes.—In the convalescent homes for patients from the New York hospitals there were treated during the year 77 men and 74 women, to whom were furnished 2262 days' board, at an estimated cost of one dollar per day. This number of patients is 33 in excess of the number treated the preceding year, showing that this summer provision for convalescent patients from general and surgical diseases, who have recently been under treatment in the New York hospitals, is much appreciated by those needing it, and contributes essentially to the comfort and recovery of quite a large number of recently sick people, and it is being more and more recognized by both physicians and patients as an important part of the New York hospital facilities.

During the coming year it is expected that a new fire-proof cottage for a few remunerative patients will be added to the Bloomingdale facilities in caring for such cases, it being the result of a legacy to the hospital to erect a Memorial Cottage to the late James H. Banker, which will be available within a few months.

—*Utica State Hospital, Utica.*—Under the direction of Dr. Adolf Meyer, of the Pathological Institute, a special impetus has been given to the medical work of the hospital. Members of the staff have attended courses in psychiatry at the institute and have put in operation new methods of procedure in the examination and treatment of cases and the keeping of records. The individual attention to patients and the thoroughness with which examinations are made is evidence of increased interest on the part of the physicians and augurs well for the benefit of the patients.

The new central kitchen is nearly completed. All the cooking for the patients of the main building will be done here. The old kitchens in the basement will be vacated. The men's department has had new plumbing installed throughout and the women's division has been re-wired. A water tower and tank with a capacity of fifteen thousand gallons has been erected to increase the water pressure in case of fire. A new chemical engine, fire alarm box, and a number of extinguishers have also been added.

—*Willard State Hospital, Willard.*—There has been no material change in the organization or management of the hospital since October, though

there have been many additions to the equipment which will increase the general efficiency of the institution. In this connection it might be mentioned that a new Hagan mangle has recently been installed in the laundry; several rooms at Sunnycroft (formerly known as D. B. 3) have been added to and raised an additional story; the third floor of the hotel has been remodeled and equipped to furnish accommodations for a number of married employees; and there has been recently purchased a combined traction engine, road roller and wood-sawing outfit, which can also be used for several other purposes and will be of great assistance in the general work of the farm.

The effort to improve the sanitary condition of the buildings occupied by patients continues. At present work is progressing on new plumbing in one of the groups of buildings, and it is hoped to extend this work so that all the buildings on the grounds will soon be provided with new and sanitary plumbing. A number of the buildings have within the past year been thoroughly cleaned and disinfected and then painted. This work is still in progress, and it is also the intention to push the work in this direction until all the buildings are overhauled. Despite the improvement in the sanitary condition and the continuance of every effort to suppress the diphtheria epidemic, cases of this kind continue to occur at times. During the past calendar year there were fifteen actual membrane cases, but no deaths. With this number of clinical cases of diphtheria there were necessarily a considerable number of germ cases which were quarantined. Immunization and quarantine, together with efforts to improve the general sanitary condition of the institution, and strict disinfection, are the measures used at present to combat the disease. An epidemic of mumps began early in December and many cases have occurred since. Employees have been affected by this disease to a much greater extent than patients. This has also been true of diphtheria of late, and it has been noticed that the newer employees were the persons most liable to be affected. Cases of erysipelas continue to occur at times, though not with the same frequency as formerly, and it is believed that the betterment in the sanitary conditions has been responsible for the diminution of cases. During the past winter influenza has been prevalent, particularly among the employees, and the large number of infectious diseases among the paid employees has crippled the force greatly. It is only by the employment of temporary attendants permitted by the State Commission in Lunacy that the outside work necessary in an institution of this kind has been carried on, and at the same time the necessary care given to the acute insane. The prevalence of epidemic diseases has necessarily occupied much of the time and attention of the staff, which would otherwise have been given more particularly to purely psychiatric work. The modern methods in this latter work have been taken up quite eagerly by the staff, and, despite the vacancies on the staff itself, each case admitted has been carefully studied along the lines suggested by Dr. Adolf Meyer of the Pathological Institute. Two of the physicians have taken courses of instruction in the Pathological Institute during the past few months, and after the present vacancies on the

staff are filled, and the demands on the time of the physicians, due to epidemic diseases, are lessened, it is hoped to prosecute the newer methods along psychiatric lines with increased energy.

The hospital acquired an adjoining farm of ninety-seven acres in September, 1903, and much work has been done during the winter in clearing the land, making roads, etc., as well as in enlarging and altering the farm house for the accommodation of thirty patients, whom it is proposed to locate there. This farm is a very valuable and desirable addition to the farm lands of the hospital, and the remodeled farm building will be a very suitable place for a farm colony in connection with a hospital of this kind.

The old names of the buildings were changed with the consent of the State Commission in Lunacy in January last, and the present names are stated herewith: Main Building, "Chapin House"; D. B. 1, "The Maples"; D. B. 2, "The Pines"; D. B. 3, "Sunnycroft"; D. B. 4, "Edgemere"; Infirmary, "The Hermitage"; Branch, "Grand View"; Superintendent's House, "Brookside"; North Cottage, "Lake Farm"; South Cottage, "Vinelands"; Steward's House, "Bleak House"; Meddick House, "Hillside"; Farmer's House, "The Grange."

—*Buffalo State Hospital, Buffalo.*—Contracts are being drawn for four new buildings at the Buffalo State Hospital, comprising the superintendent's residence, residence for the medical staff, chapel and amusement-hall, and a home for men nurses, to accommodate one hundred persons—the total amount being in the neighborhood of $93,000. These buildings will increase the capacity by allowing quarters now occupied for other purposes to be used for patients.

—*Hudson River State Hospital, Poughkeepsie.*—An unusual incident occurred one night in August last, when Frank Sanders, a night attendant, became intoxicated and assaulted two patients. His arrest was caused by the hospital authorities and he was afterward indicted by the grand jury, and served five months in jail.

Clinical staff meetings are now held on Mondays and Thursdays weekly. Matters concerning hospital routine and administrative questions touching discipline, etc., are given attention at special meetings, so that the work on the days above mentioned is purely medical in character. Each new patient is presented to the members of the staff, and the physician who has charge of the patient makes a thorough mental as well as physical examination before the staff. Thus the meeting is practically a clinic. Much benefit is derived from the discussion of cases.

Statistical Studies.—Of the 9735 patients admitted from October 1, 1878, to October 1, 1903, it is found that 544 men and 77 women suffered from general paresis, while 1091 men and 1015 women were cases of terminal dementia. On the remaining 7008, 530 were epileptics, 1950 were diagnosed mania acute, 90 as delirous mania, 800 mania chronic, 2080 melancholia acute, 782 melancholia chronic, 180 as paranoia, 431 as imbecility, 90 as primary dementia, 75 as not insane.

—*Report of Middletown State Homeopathic Hospital, Middletown.*—The past year at the Middletown State Homeopathic Hospital has been, on the medical side, marked by a quickening of interest in the study of the clinical types of insanity. The case histories have been taken in much greater detail than ever before, accurate records of the mental and physical conditions of the patients upon admission made and the development of their psychoses watched and recorded. The classification of cases for study according to the methods of Kraepelin has given a new impetus to the analysis of mental symptoms, has permitted their arrangement into more natural groups and has shown a considerable number of psychoses demanding further study. Care has been taken by correspondence and conversation with the friends of patients to determine, as far as possible, the family predisposition, etiological factors and the circumstances of the onset; also the patient's ability, temperament, etc., in health, the nature of previous attacks of mental disease, if any, and his condition in the intervals. In this way a great deal of instructive material not readily lending itself to tabulation has been passed in review in the staff meetings.

Staff meetings are held daily except Sunday. The physicians, besides consulting on the condition of the sick, make informal reports of the symptoms and progress of interesting cases in the several services; discuss the condition of patients about to be discharged, and, as a rule, one new patient's history is read at each meeting in detail. Following this, if his condition will permit it, the patient is presented and doubtful points are cleared up as far as possible by additional examination; treatment is discussed and an effort made to arrive at a diagnosis and prognosis.

Dr. George F. Brewster, of the hospital staff, is spending the winter in New York in study at the Pathological Institute.

In all instances where consent could be obtained autopsies have been performed on the remains of patients dying in the hospital. One less than half of the 53 patients dying in the hospital for the year ending September 30, 1903, reached the post-mortem room. In this connection the hospital has benefited by the New York State Pathological Institute at Ward's Island, New York City, under direction of Dr. Adolf Meyer, where pathological material sent has received expert study, reports and drawings of the specimens made and duplicates of sections cut are promised for the hospital.

Next summer it is proposed to build a separate pavilion for the accommodation of one hundred cases of tuberculosis with verandas for out-of-door treatment and a separate kitchen and dietary. This will permit the better treatment of consumptives and relieve the wards of these patients.

The contract has been let for a surgical and clinical pavilion. It will contain on the first floor, besides an operating room, a room for instruments, sterilizing room, etherizing room, gynæcological room, a room for electro-therapeutics and diagnosis and a bath-room. Part of the building is to be two stories high, and on the second floor will be five sleeping rooms and a bath.

Another addition contemplated is one of the several pavilions for sixty acute insane, each, to be located on the grounds of the existing State hospitals and so distributed as to give all portions of the State facilities for the better care of possibly curable cases.

Turning to improvements actually accomplished, the year has seen two pavilions containing 554 patients made much safer from danger of fire by the removal of old and defective electric wiring put in before present-day methods were in use, and the substitution of more heavily insulated cables in concealed iron pipes. The floors of the bath and toilet sections of these pavilions have, because supported only by decaying wooden beams, been torn out and replaced by tile floors supported on steel girders and concrete. In these rooms antiquated plumbing has been replaced by new fixtures of modern design.

During the past summer the pond on the grounds has been dug out and its margins walled with stone. During the winter an abundance of ice has been secured, rendering the institution independent of its previous source of supply. This work has been done by patients and employees. A new and larger ice house has been built in the same manner.

A beginning has been made in the accumulation of a museum of patients' handiwork; a room set apart for the reception of striking and original articles, and it is believed that by adding to it from time to time an interesting and instructive collection can be gathered.

A very pleasant dining-room has been made by transforming a light and commodious portion of the basement under Talcott Hall. It accommodates the patients in two nearby cottages, thus adding eight new beds, besides diminishing the administrative cost by rendering unnecessary the maintenance of a separate kitchen.

A stone-crusher run by the same portable engine used to furnish power for the ensilage cutter has been purchased and installed because of the exhaustion of the natural gravel beds on the farm.

Fourteen electric fans have been placed in all the hospital wards, adding to the comfort of the patients and improving the ventilation.

A new, more extensive, and more efficient telephone system has been installed, connecting each ward with the central station in the main office.

The circular Custodis chimney is now completing its second winter's service and gives excellent satisfaction in every respect.

—*Manhattan State Hospital, West, Ward's Island, New York City.*— Clinical work in all departments has continued with undiminished interest. Great care is exercised in the study of each case as admitted, typewritten notes are made, with additions thereto from time to time, detailing the patient's mental and physical condition and the treatment of the case, and afterward, each case is assigned to some physician for analysis, which is presented for discussion at the usual Friday afternoon staff meeting. If doubt is entertained in regard to the classification of any particular case, it is reconsidered at some future meeting, usually the Tuesday evening meeting, which is supplementary to the regular Friday afternoon meeting,

and the case is again brought forward for classification. Written summaries are presented in each case and the reasons for the author's conclusions are given therein. In this connection, interest in pathological work is constantly developing, and autopsies are held by the autopsy physician whenever consent of the relatives can be obtained.

Gynæcological work continues under the supervision of Dr. Le Roy Broun, assisted by Dr. Rawls and the physicians of the hospital service. The work done in this department has given excellent results and much permanent good is derived therefrom.

The work under the supervision of Dr. William H. Thomson among the epileptics has been continued by Drs. Robert C. Kemp, Graham Rogers and John E. Traub. Dr. Achilles Rose has also become interested in this department and visits the hospital regularly, giving his advice and the results of his experience.

Dr. Ferd. C. Valentine continues regularly to carry on the work in his department, namely, genito-urinary diseases. Favorable results have been obtained by him in the treatment of enuresis among the insane by the epidural injection of deci-normal salt solution.

Special attention has been given to gastroptosia among the epileptics by Drs. Kemp, Rose, Rogers and Traub, and a chart showing the location of the stomach in 27 cases has been prepared by Dr. Kemp. This chart, prepared after trans-illumination of the stomachs in these cases, indicates that the stomach is in normal position in only 2 cases out of the 27.

Dr. Lusk has always promptly responded to calls when his services as general surgeon have been required.

Drs. Ward A. Holden, ophthalmologist, and Henry H. Whitehouse, dermatologist, have been visiting the hospital regularly.

In regard to the phthisical service, no special changes have been made since the last summary was issued.

The tents for acute cases referred to in the last summary were continued in use until late in the fall, when, as cold weather came on, they were vacated and the patients returned to the wards. The results in the care of these acute cases in the tents have been so gratifying that it is intended to continue their use during the coming summer season.

A new solarium ward, 115 feet long and 19 feet wide, intended to accommodate about 50 patients, has been erected to adjoin the east end of Ward 21—the ward for old, feeble and bed-ridden patients. This not only increases the capacity of the hospital as a whole, but it affords a very pleasant and comfortable ward for the use of this class of patients.

The usual amusements have been continued during the past winter. The employees gave four very interesting entertainments for the patients, and Mr. W. H. McCollin, the blind entertainer, made his annual visit and gave an entertainment for the patients. The orchestra continues in good training and gives, regularly, band concerts on the wards during the winter season.

One of the physicians has been detailed to attend the three months' course of instruction at the Pathological Institute, where a class of six, selected

from the various hospitals, has been attending during the current quarter. The clinical work by these gentlemen is carried on in the various wards of the hospital, Ward 27 having been especially set apart for the use of the Pathological Institute.

—*Gowanda State Homeopathic Hospital, Gowanda.*—This hospital is at the present time advertising for bids for four buildings, namely: a superintendent's residence, a staff house, a nurses' home and an amusement hall. This will take the staff and superintendent out of the administration building and provide for about 100 additional patients. The present census is 700. Plans have been submitted to the State Commission in Lunacy, at their request, for a new hospital for the acute insane to accommodate 60 patients. There has also been completed a new water-works system from springs situated on the Cattaraugus Indian Reservation. These springs will supply us in the neighborhood of 300,000 gallons of water a day. The process is the Bacon air-lift system.

—*The Long Island Home, Amityville.*—The Long Island Home reports the proposed extension of the east wing, female halls, for ten additional patients, dining-room and nurses' quarters, carrying out the same structure as the present main building to which it is to be an addition, with sun-parlors; also the installation of a static-electric machine, with X-ray outfit.

—*The Craig Colony for Epileptics, Sonyea.—Improvements.*—Two large infirmaries with a capacity of 125 to 150 beds each will shortly be opened for feeble and disturbed cases. Any large colony or institution for epileptics accumulates in time a large number of such cases. Heretofore at Sonyea these have been cared for in small cottages. The new buildings about to be opened will permit their segregation in two groups—one for men and one for women. Twenty per cent of the 1800 epileptics now under care in the German Colony at Bielefeld are of this class. Ultimately about the same per cent of all the patients at the Craig Colony will live in the two infirmaries.

Contracts have just been let for two new cottages in the women's group, each holding 20 patients of the better class; an addition to the Peterson Hospital in which there is provided a complete hydrotherapy equipment, a medical library room, and rooms for a dispensary for outdoor patients; an addition to the laundry, and another filter bed for the sewerage system. The two cottages will cost $28,000; the hospital addition, $16,000, and the sewerage bed, $2000.

Colony Model at St. Louis.—A model of the Craig Colony has been made by Mr. Francis T. Gilling, of Shokan, Ulster County, New York, for the St. Louis Exposition. The model embraces some 38 to 40 houses, showing sections of the Colony only—the village green in the division for men and the Villa Flora group in the division for women. The great size of the Colony plant made it impracticable to reproduce it on a scale large enough to give satisfactory results. The exhibit will be in the Educational Building in the Division of Social Economy.